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THE PAROXYSMAL HÆMOGLOBINURIA OF HORSES AND CATTLE AND OF MAN.

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WHILE looking through veterinary literature for another purpose, I became interested in a variously named affection of horses. The names given are azoturia, hæmoglobinæmia, *hæmoglobinæmia paralytica*, hæmoglobinuria, hæmoglobinuric toxæmia, lumbago and black dysuria. A similar disease occurs in cattle. It is a paroxysmal affection and in this and in some other respects it seemed to share features in common with the condition described in text-books of human medicine as paroxysmal hæmoglobinuria, but in other respects it seemed to be different.

The disease has received no satisfactory explanation and unsolved problems challenge the inquirer. Thus induced, I have given thought to the subject and I venture here to present my ideas. Time and more inquiry will reveal to what degree I have been successful. I do not pretend to have done more than present some plausible-looking theories and I cannot afford to be dogmatic. Everything is provisional and in any case I leave a good deal unexplained. But what there is may well go into the forum of debate, for it is an interesting problem.

Azoturia of Horses.

As regards azoturia in horses, the matter following is an abstract from the account given by Friedberger and Fröhner[1] who have written about the disease at considerable length. The abstract is for the most part in the exact words of Friedberger and Fröhner, but non-essential matter has been left out.

The symptoms are as follows: In most cases the animals, having stood idly and well-fed in their stalls for some days, where the air has been warm and ventilation bad, on returning to work shortly begin to manifest irregularities in their movements, usually within a quarter to half an hour after starting. In mild cases the symptoms suggest rheumatism. The animals are stiff or even lame, in the hind limbs especially. At the same time partial sweating breaks out. In severer cases the horses stagger on their hind legs, have a stiff and insecure gait, there is laboured propulsion of the hind limbs, the angles of the joints remain more widely open than usual so that the whole limb seems longer and the hoofs are dragged on the ground. Then the animals knuckle over at the pastern and tremble, sweat, have difficulty in preserving the equilibrium of their hind limbs and not infrequently fall down as though struck by apoplexy. When on the ground they make frantic attempts to rise, show signs of difficulty of breathing and of terror, and are occasionally bathed in sweat. Occasionally more localized symptoms are observed and not necessarily in the hind limbs only.

Another striking symptom is the hæmoglobinuria which appears in all severer stages of the malady and which was formerly considered an invariable sign of the disease. But hæmoglobin may be absent from the urine in mild cases or in those with abortive course. The urine which contains hæmoglobin or methæmoglobin, is characterized by its dark red, at times ruby red colour or varies from a dirty brown to an almost inky black colour.

According to French practitioners, the urine should contain sugar.

There is usually no rise of internal temperature. The visible mucous membranes are usually redder and of a dirty colour from solution of hæmoglobin. In the digestive organs the diminution or even suppression of peristalsis is in many cases very noticeable. The sensorium is unaffected and disturbance only occurs when there is complication with nephritis, when there may be epileptiform spasms.

The course is generally acute. Recovery in mild abortive cases may take place in an hour or two or within two or three days, in which case it is often a complete one. We must not forget, however, that the course of this disease is occasionally marked by periodical increases of violence and after apparent improvement relapses may occur. For instance, a horse may fall several times a year if kept a few days in its stall. Occasionally the same animal will be ill several times in a few weeks. A simultaneous affection of several horses in the same stable has also been observed. In severe cases recovery often leaves a paralytic condition of the extremities behind. When the sickness ends in death, the paretie conditions grow more and more intense and pass finally into complete paralysis. The animal gets more restless, breathing becomes dyspnœic and decubitus constant.

Cases are separated into two forms. Fröhner's "rheumatic" hæmoglobinæmia is regarded as an inflammation of the muscles of the hind limbs produced by cold, whereby a dissolution of the muscular colouring matter (identical with hæmoglobin) and its passage into the blood takes place. Those rarer cases which are brought on by over-exertion, as they regard parenchymatous myositis, and in contra-distinction to "rheumatic" they are described as "spontaneous" hæmoglobinæmia.

"Rheumatic" Hæmoglobinæmia.

The commonest cause of rheumatic hæmoglobinæmia is probably a preceding chill. The animals acquire a predisposition to it by standing some days in warm, steamy and ill-ventilated stables, being well-fed the while, for instance during holidays or when lame. The warm and well-protected stables of the richer owners are proportionately more troubled with the disease than the badly-appointed ones of the poorer classes.

"Spontaneous" Hæmoglobinæmia.

After excessive muscular action, a severe, acute muscular degeneration, especially of the psoas muscle, the *longissimus dorsi,* the gluteus, the *quadriceps femoris* and the anconeus is frequently observed. It may be general, or confined to a single group of muscles and may occur on one or both sides, and may or may not be accompanied by hæmoglobinuria. Respecting symptoms, course and *post mortem* appearances, this "spontaneous" hæmoglobinæmia is the exact counterpart of the "rheumatic" form.

In a few cases the agency of chill appears to be absent. For lack of a more definitely known cause, these may be termed "infectious," that is "toxæmic" hæmoglobinæmia.

Chill is acknowledged by the great majority of observers to be the chief cause of the disorder.

Post Mortem Conditions.

The chief changes found *post mortem* are in the muscles and blood. The muscular changes which are seen chiefly in the muscles of the croup and thigh, consist to the naked eye of œdematous swelling and paleness. Microscopically they show granular opaqueness, a splitting up into layers, hyaline degeneration and loss of transverse striation. Occasionally small hæmorrhagic foci are seen. The most important changes in the blood are loss of normal colour and an appearance resembling varnish or tar and no coagulation, or only slight coagulation.

Other Changes.

As regards glycosuria, Law[2] in his text-book of veterinary medicine under "Glycosuria" remarks: Rueff and Mouquet each contribute a case occurring in paraplegia attendant on hæmoglobinuria and in which the amounts of sugar were respectively 5.85% and in 1.01%.

Paroxysmal Hæmoglobinuria in Cattle.

According to Friedberger and Fröhner hæmoglobinuria originates in cattle, as in horses, from chill. It usually occurs in cold, wet, stormy seasons, when animals are driven from sheltered quarters too early for pasture, while it does not appear if they are turned out later in the season. It may be brought on by drinking water which is too cold. This corresponds to their "rheumatic" form. The authors quote many cases of hæmoglobinuria in draught oxen exactly similar to those occurring in working horses. It occurred practically always in oxen put to work in cold inclement weather after a period of rest in the stable. It seems that in all forms there is a degenerative change found in the muscles and especially in the buttock, psoas, thigh and shoulder muscles.

In New Zealand, J. A. Gilruth[3] has recorded under the names of red-water and hæmo-albuminuria a condition apparently identical with the disease in cattle as described by Friedberger and Fröhner, though a description of the condition of the muscles is not supplied. It occurred elsewhere, but it was especially noted on some dairy farms in the county of Southland, at the extreme south end of New Zealand and the coldest, wettest and bleakest county of the Dominion. According to Gilruth it was always associated with feeding injudiciously on turnips. About the commencement of spring the cows were simply turned out on to the paddocks of turnips with a limited supply of straw and were unhoused at night. The turnips were at freezing point and the animals gorged themselves on this frosty food. Under these circumstances one farmer lost seven of his herd from the disease. These cows were near calving.

In a later report Gilruth[4] records the result of a microscopical examination of specimens of liver and kidney from a cow which had contracted the disease while feeding on a field of turnips during wet weather. The following conditions were shown:

Liver: Large areas of cloudy swelling, with only a very small area of each lobule, situated around the intra-

lobular vein, normal. While the nuclei of the healthy cells took nuclear stains well, those of the degenerated cells stained but faintly. Kidney: Glomeruli swollen (cloudy swelling), so much so that the usual space between these and Bowman's capsule was obliterated. The cells lining the convoluted tubules were swollen and ragged in appearance on the free edges, whilst many of the tubules contained a greenish-yellow colloid-looking material.

For a special reason that will appear in due course I will not enter here into the consideration of the paroxysmal hæmoglobinuria of man, but will proceed to discuss the equine and bovine cases as if they were alone to be dealt with. Before doing so, however, it will be desirable to consider certain physiological matter.

Catalase.

We turn our attention, therefore, to catalase, an enzyme upon which there has grown up in the past twenty-three years a copious literature. I shall make use principally of the work of W. E. Burge and his associates. Burge has made a special and prolonged study of catalase and has produced a very large number of papers thereon.

Catalase is found in all actively metabolic tissues of plants and animals, from the lowest to the highest, from bacteria (anaerobes excepted) to man and evidently plays an important part in the activity of organized life. Its particular enzymic rôle consists in the decomposition of peroxide of hydrogen with liberation of nascent oxygen. It is obvious that it is intimately concerned with tissue oxidations and Alvarez and Starkweather[5] remark: "It seems probable that in some way the catalase assists in furnishing oxygen to the tissues as fast as they require it." According to W. E. Burge and A. J. Neill,[6] it is issued to the systemic circulation from the liver, where it is principally formed, though it is also formed in the gastric and intestinal mucosa and perhaps also in the pancreas and spleen. The liver is very rich in catalase and according to W. E. Burge[7] the catalase content of the blood can be increased by electrical stimulation of the splanchnic nerves distributed to the liver. J. Kennedy and W. E. Burge[8] found that in depancreatized dogs the catalase content of the liver was decreased by about 75% resulting in a decreased output of catalase into the blood. They suggested that this might mean that the pancreas gives off an internal secretion which is carried to the liver and increases the formation of catalase in this organ. Of course this is not the only possible explanation. Catalase is destroyed (like the oxidases) in the processes associated with its activity and is therefore constantly disappearing at the seat of oxidation.[6] It appears that this destruction is wrought by the nascent oxygen which the catalase itself produces. Nascent oxygen, however, is not merely destructive to catalase. It is able to destroy all the ordinary enzymes, as is shown, amongst others, by W. E. Burge and E. L. Burge,[9] using platinum black. The paper just referred to in which this property of nascent oxygen is considered, is for my present purpose a very important one and it will be necessary for readers to make themselves personally acquainted with it. Since the original will be beyond the reach of most, I have taken the liberty of making a rather long extract. Its application will be considered later.

The fact that pepsin and trypsin are easily oxidized and that the mucosa of the stomach and intestine possesses intense oxidative properties would seem to offer an explanation of the fact that these organs are not digested by the pepsin and trypsin contained within their lumen. The assumption would be that the pepsin and trypsin immediately in contact with the mucosa of the stomach and intestine respectively undergo oxidation and that by such means the cells maintain their integrity during life. Salkowski and others (*Deutsche Klinik*, 1903, II., 147) have shown that all the body tissues possess the power of undergoing autolysis after death and that under certain normal as well as pathological conditions tissues and even organs may undergo autolysis during life. The atrophy of the thymus and the involution of the puerperal uterus might be mentioned as examples of normal auto-digestion. Various theories have been advanced to account for the fact that the tissues do not undergo auto-digestion during life as after death. One theory (Glassner, *Hofmeister's Beitrage*, 1904, IV., 79) is that there are in the living tissues anti-substances which hold the autolytic enzymes in check. Another theory (Wiener, *Centralblatt für Physiologie*, 1905, XIX., 349) suggests that the tissues are protected by their alkaline reaction, as it has been shown that an acid reaction is necessary for the activity of autolytic enzymes. A third theory assumes that the enzymes exist in a zymogen form and are activated or inactivated as the need may arise.

In view of the fact that autolytic enzymes in common with all the ordinary enzymes are destroyed by nascent oxygen, an additional theory may be advanced, namely that the tissues maintain their integrity during life by means of their oxidative properties. This theory would assume that normally a balance exists between the autolytic enzymes and the oxidative processes of the tissues. It is known that in infectious diseases (Flexner, *University of Pennsylvania Bulletin*, July, 1903), in diseases of the circulatory and respiratory systems (Schlesinger, *Hofmeister's Beiträge*, 1904, IV., 87.), in acute yellow atrophy of the liver and in chloroform and phosphorus poisoning autolysis may be increased to a marked degree. Without the oxygen continually supplied by the circulatory and respiratory systems oxidation in the tissues would be impossible. Since this is true, any special interference with either of these systems would presumably result in a decreased oxidation in the tissues. If the balance which has been assumed to exist between the oxidative and autolytic processes, exists, then any interference with the supply of oxygen to the tissues should express itself in an increased rate of autolysis. Schlesinger (*loco citato*) noted an intense self-digesting tendency of the tissues in diseases of the circulatory and respiratory systems. Under such conditions the amount of oxygen supplied to the tissues is decreased and the fact that under these conditions autolysis is increased would seem to support the above assumption. Jacoby (*Zeitschrift für Physiologische Chemie*, 1900, XXX., 174) showed that the livers of dogs dead of phosphorus poisoning underwent autolysis more rapidly than normal livers, while Welsch (*Archives Internationales Pharmacodynamie et de Therapie*, 1905, XIV., 211) and Riess (*Berliner Klinische Wochenschrift*, 1905 (42), 44A, 54) found that oxidation in cases of this poisoning is decreased. Welsch made a study of the respiratory exchange in cases of phosphorus poisoning and found that the oxidative processes were decreased by about 20%. Riess proved the deficiency of oxidation also by showing the presence in the urine of large amounts of organic acids which are oxidized under normal conditions.

In regard to phosphorus poisoning, W. E. Burge[10] fed cats on food containing yellow phosphorus for varying periods and studied the effect on the catalase content of the liver, heart and blood. In the livers of animals that had eaten phosphorus

for three days, the catalase was diminished 23%, in those poisoned for six days it was diminished 60%. In the heart it was diminished 17% and 27% respectively. In the blood there was practically no decrease after three days, but there was 28% decrease after six days. The livers of the animals that had been fed three days, presented the typical appearance of fatty degeneration with little or no indication of autolysis, while the livers of those that had been fed six days, showed extreme autolysis as well as fatty degeneration. The livers of these severely poisoned animals were literally in a state of falling to pieces as a result of autodigestion. From this it seemed that the amount of autolysis was inversely proportional to the amount of oxidation.

Another paper by W. E. Burge supplies us with further important data.[11] He gives his conclusions as follows:

1. The amount of catalase in the different muscles of the body varies with the amount of work done by these muscles; those doing the greatest amount of work contain most catalase, while the muscles doing the least work contain least catalase.
2. By increasing or decreasing the external physical work of a muscle the amount of catalase is correspondingly increased or decreased. If the external physical work of a muscle such as the pectoralis of pigeons is reduced practically to zero the amount of catalase is reduced by approximately 38%.
3. Catalase is greater in amount in the muscles of warm-blooded animals in which oxidation is more intense than in corresponding muscles of cold-blooded animals in which oxidation is less intense.

Could the Equine Disease be Explained in Terms of Catalase Deficiency?

In warm-blooded animals the general metabolic processes of the body are adapted to carry on at or very near to a certain level of temperature which varies somewhat in different species. The organism possesses means of regulating the balance between heat production and heat loss so that an even temperature may be maintained in its vital parts. The mechanism is partly chemical and partly physical. The chemical regulation is procured by alteration of the rate of combustion of fuel, more being burnt as required when heat loss is great, whereas when the surroundings are warm, combustion is reduced to a minimum. The seat of this process of combustion is in the muscles. They are the chief furnaces of the body and it is to be remarked that though combustion is active in a muscle doing mechanical work, it is also active whenever required for heat production, whether work is being done by the muscle or not.

The physical regulation is achieved by increasing or decreasing the quantity of heat lost. The chief channel of heat loss is the skin. When the surroundings are warm and when it is necessary to get rid of a large amount of heat, the skin becomes warm and flushed with blood and in animals that sweat, sweat secretion is poured out. If while adjusted to such warm surroundings an animal is suddenly exposed to cold and especially to cold wind or to cold rain or fog, there immediately ensues a great flood of heat loss and this continues until the body becomes re-adjusted. Naturally this tends to produce a fall in the level of temperature. The physical mode of re-adjustment is by altering the condition of the skin which from being warm and flushed and moist becomes cold, pale, shrunken and dry. At the same time the mechanism of chemical regulation is put to work. The rate of combustion is forced up in the muscles and more heat is produced as compensation for the increased loss that has been and is being incurred. This process, therefore, involves a "rush" call on oxidation, a rush on the stock of fuel and a rush on the stock of catalase in the muscle.

In the stable the heart and respiratory muscles are at constant work, but the muscles of the limbs are confined to the work done in supporting the weight of the animal. If Burge's observations have any application, they indicate that the catalase content of the muscles of the limbs will be adjusted to the metabolic requirements of supporting the weight only and not of progression, as in the cognate case of the pectoral muscle of pigeons. The catalase content of the muscles of the hind limbs will not need to be any greater than it is in the fore limbs. It is quite otherwise, however, in progression. In progression the great thrusting muscles of the hind quarters do the hard work, the muscles of the fore quarters not being concerned in the thrusting, but in subsidiary action. When a horse is harnessed to a load and driven, there is at once a call for power and the motor stimulus activates the metabolic apparatus in the thrusting muscles. Oxidation increases, catalase is requisitioned, the present supply is used up and more is demanded. Under normal conditions the demand is supplied. But suppose there is something wrong with the liver function, be it temporary or permanent, so that it is inadequate to supply catalase to the full extent required. In that case though the motor stimulus to the muscle continues, the catalase content gradually fails. With the disappearance of the catalase the protective physiological inhibition of the local digestive enzymes disappears also.

There is another accompanying factor. It is known that in the processes by which energy is liberated in the muscles, there is in the intermediate metabolism a formation of lactic acid. Normally this does not accumulate. It has been shown, however, by Fletcher and Hopkins[12] that when there is a deficiency of available oxygen in the muscle, lactic acid accumulates. Presumably, an inadequate supply of catalase at the seat of activity would reduce oxidation and so favour hæmolysis and autolysis. Autolysis requires an acid medium. In the absence of oxidation, therefore, there ensues paralysis of function, accumulation of acid and autolysis. Thus the structure of the muscle is destroyed. If this can occur, the blood passing through the muscle is also exposed to attack, at least to some degree, and its cellular elements will therefore become disorganized. In this way an hæmolysis as well as autolysis of muscle may be presumed to occur at the seat of the lesion and from that source the product enters the blood-stream and passes to the kidneys, where it is excreted.

The presence of sugar in the urine could reasonably be accounted for by liberation of glucose by hydrolysis of glycogen in obedience to the motor requirements, but inability to oxidize this sugar in the great muscles to a parallel extent causes an accumulation. The raising of the glucose content of the blood then produces the glycosuria.

I have said that during rest in the stable the heart and respiratory muscles of the horse are continuously at work and I contrasted this with the condition of the thrusting muscles. Nevertheless, during exercise the heart and respiratory muscles have to increase their output of work very greatly, though the contrast is not like that of the thrusting muscles which pass from prolonged rest to extreme activity in a few seconds. I am prompted here to turn aside for a moment to refer to the phenomenon of "second wind" is athletes. During violent exercise there is after a time considerable dyspnœa, but if the exercise be continued the discomfort disappears sometimes quite suddenly. Pembrey[13] has stated that the physiological explanation is unknown. I would offer the suggestion that "second wind" might be plausibly explained by imagining that it is brought about by the establishment of catalase equilibrium in the heart and respiratory muscles, catalase production having caught up to the demand.

Possibly one could explain the pain and stiffness caused by over-use of an untrained muscle by imagining a minor degree of what we have supposed to happen in the thrusting muscles of a horse whose catalase production is deficient.

It may be said that I have no right to suppose a derangement of liver function merely at random and no better ground than that it provides a way out of a difficulty. But it is not a random throw. There is more ground for suspicion than many might expect. There is nothing clear, nothing definite, but there is something to think about.

There is a special peculiarity of the horse that has frequently attracted the attention of observers, and that is its liability to various disturbances of the digestive apparatus. The horse is very subject to complaints causing abdominal pain—a symptom referred to generically as "colic." Accompanying the colic there is very often muscular incoordination in the limbs, a symptom known as "staggers." Motor irregularities giving rise to tympanic distension are common in the stomach and further along there is a propensity to volvulus and intussusception. If a horse is driven soon after a hearty feed he is particularly liable to show symptoms of acute indigestion, associated with gastric tympany. Flatulent distension of the stomach after food is common enough in the human subject, sometimes accompanied by vertigo, which doubtless corresponds to the "staggers" observed in animals. Other reflex effects are common in the human subject, especially within the area of distribution of the autonomic fibres of the vagus. Of these palpitation is one of the most obvious. But in certain predisposed persons, namely people with biliary calculi, dietary indiscretions may bring on an attack of biliary colic. The reflex has then affected a part of the hepatic apparatus that we can recognize by familiar clinical effects. But we have no right to assume that the liver itself never suffers a reflex disturbance because our often limited diagnostic facilities do not make it conspicuous. On the contrary, we should be rather surprised if the liver were immune. Indeed, writers on human medicine say that acute congestion of the liver is liable to follow or accompany digestive disturbances located primarily in the stomach. Is the liver immune in the horse? I find that writers on veterinary medicine do not think so, for they also describe hepatic congestion or disorder of hepatic function as liable to be excited by gastric or gastro-intestinal disturbances. But there would be various grades of disorder and not every disturbance of function would be conspicuous. Sometimes such disturbances would only be recognizable by bio-chemical tests not readily available or not often applied. It is at least conceivable that a defective response to the demand for catalase might be one of those bio-chemical effects and this inadequacy might only be great enough to become openly recognizable under special circumstances, for example when there has been a great and sudden heat loss combined with or immediately followed by a great general demand *plus* a superlatively great and concentrated demand in a particular portion of the muscular system. Then bankruptcy ensues. A chronic or permanent disability of the liver might be present and only become evident in times of special stress, the liver being equal to ordinary requirements.

Let us now consider another possibility, namely the effect of a chill, that is a flood of heat loss, not only on liver function, but also on general physiological efficiency. Compensatory adjustments are not immediate and in individuals untrained to sudden changes or in those whose physiological efficiency is already hampered by some food fault such as inadequate vitamin or amino-acid supply or by mineral shortage, adjustment may well be more difficult than in those trained to quick adaptation. In any case, in cold or inclement weather a sudden exposure while the skin is warm and flushed must involve a very great heat loss. Does this chilling itself lower the functional efficiency for a time of the liver and other parts of the digestive apparatus? Whether that be so or not, it does seem temporarily to lessen resistance to some infections, at least in human beings, for it is a chill after exposure to infection (say in a theatre), that seems often to be the cause of "catching cold."

In this connexion we may also note the popular belief that it is injurious to indulge in a cold bath or to go swimming immediately after a hearty meal. This opinion seems on the whole to be endorsed by the medical profession. Why is it endorsed? If it be said that it is liable to cause depression of digestive function, shall we arbitrarily limit the depression to one function or one group of functions or to one part of the function of the liver or of the pancreas or of the intestinal epithelium? If there be a depression of function, it would be either

the heat loss or the increased metabolism following the heat loss that would exercise the detriment.

The conditions just mentioned and particularly so in the case of swimming, seem closely comparable to those of a horse taken from a warm stable after a good feed and driven in cold inclement weather.

The Fundamental Causes of the Condition.

It remains for us now to go beyond the causes that actually precipitate an attack and to find out, if we can, some reasonable theory to account for the predisposition. We have seen that sometimes several horses belonging to the same stable have developed the malady and that the same horse may suffer from repeated attacks. In a passage that I have not reproduced, Friedberger and Fröhner says that it has been observed both in a horse and in an ox on the same day and presumably on the same farm. We have seen also that it has attacked as many as seven cows on the same farm. In these last, however, no mention is made of acute degeneration of the great muscles of progression.

The liability of horses to colic and to gastric and intestinal disturbances generally has by some been ascribed to the anatomical arrangement of the digestive apparatus in that animal. But the donkey is said to be free or relatively free from this predisposition and both animals have practically the same anatomical arrangements. It seems to me much more likely that the difference is due to differences of dietary conditions.

The horse is specially fed to produce a maximum of work either by traction or by speed and energy-producing food in a concentrated form is therefore given. That is, he is given a liberal ration of grain or grain products such as bran. Moreover, many horses are fed for long periods entirely in stables and can get no fresh green food.

As a matter of fact, the stabled horse receives a diet not very unlike that of some grain-fed pigs. Energy-producing food is given to a pig to produce fat, not work, but whether the energy be used or stored, that factor of the dietary must be large. But farmers do not succeed with their pigs if they fail to compensate for certain deficiencies of grain diet. The pigs must receive in addition a sufficiency of milk, whole or separated, or a liberal quantity of green fodder, especially that of leguminous plants such as lucerne. By either or both of such means is supplied a good quality of protein and in liberal amount, good quality meaning that the protein amino-acids are proportioned in the right way for animal nutrition. By the same means there are supplied also the requisite mineral elements, especially calcium and phosphorus, and these also in liberal quantities and in such ratios as enable a right physiological balance to be sustained.

The protein element of grain is relatively small and excepting in the germ which is very minute, it is of low grade quality, for in its make-up certain amino-acid "building stones" which the animal organism requires in relatively large quantity, are poorly represented.

In regard to mineral matter the ash of grain is relatively small in quantity and contains extremely little calcium, though it contains a much larger proportion of phosphorus. The hay derived from oats and other grasses is also poor in ash. The ash of oat hay varies enormously in its content of calcium and phosphorus according to the soil in which it has been grown. One sample grown in favourable soil may contain six times as much calcium and three times as much phosphorus as another sample grown in less favourable soil. On the other hand, a leguminous hay such as lucerne contains several times as much calcium as oat hay, even at its best, and may contain fifty times as much as some specimens. The same applies in various degrees to any grass hay.

As examples of the facts I quote some analyses from various sources. Calcium oxide in grain: Maize, 0.026%; wheat, 0.051%; oats, 0.143%. Phosphorus pentoxide in grain: Maize, 0.623%; wheat, 0.808%; oats, 0.888%. Calcium oxide in peas, 0.098%; phosphorus pentoxide in peas, 0.817%. Calcium oxide in bran, 0.160%; phosphorus pentoxide in bran, 3.040%. Calcium oxide in oat straw grown in different soils: 0.085%, 0.20%, 0.35%, 0.499%; phosphorus pentoxide in oat straw grown in different soils: 0.16%, 0.37%, 0.51%. Calcium oxide in lucerne hay, 4.31%; phosphorus pentoxide in lucerne hay, 0.61%.[14, 15, 16, 17]

Pigs fed on grain without either milk or green fodder fail to thrive and eventually sicken and die. The evidence goes to show that a very large part of the trouble is due to failure in the supply or utilization of calcium or calcium and phosphorus. I forbear to quote authorities for all these statements, but there is plentiful evidence. I know the literature pretty well and I am only stating what is practically accepted and unquestioned.

I have left out the question of fat-soluble vitamin which is so slight in amount in grain, in old hay, in oilcake and in certain roots supplied to pigs and to stall-fed animals, as has been shown by Drummond and associates.[18] This vitamin is known to be relatively small in amount in the bodies of pigs not receiving green food and by analogy it is not likely to be large in the body of the completely stall-fed horse or cow.

All in all, it seems that we can say this, that according to our modern knowledge in the dietary of the stall-fed animal, especially at the end of a long winter, there are liable to be imperfections formerly unthought of and almost inconceivable.

There is positive experimental evidence, as is shown for instance by the work of Steenbock and Hart[19] on the goat, that in stall-fed animals the utilization of calcium may be detrimentally affected so that even what is supplied is inefficiently absorbed. A negative calcium balance occurs on certain types of dietary. The animal may have to use up some of its stored calcium or calcium and phosphorus for its ordinary physiological needs. The evidence that the bones function for this purpose as a bank of issue for lending calcium or phosphorus

is abundant and confirmed beyond all doubt, as is also the fact that it is in some animals a frequently recurring phenomenon, as, for instance, has been shown by Forbes and associates.[20, 21, 22, 23] I would, therefore, regard the process of osteoclasis and its result, osteoporosis, as protective. Unfavourable conditions might interfere with the free course of osteoclasis and to my mind the deprivation of fat-soluble vitamin *plus* deprivation of light in a stable might well provide such unfavourable conditions. This explanation, however, is not vital to the main issue and some other explanation may turn out to be better. The main point is whether detriment may come by way of the dietary, a detriment such that we may reasonably expect important organs to become disordered easily and so be relatively unprepared for serious and sudden stresses.

All the same, it will be profitable to direct attention to osteoporosis in the horse. By using the term osteoporosis just now, I did not mean that the degree of it would usually extend to more than what could legitimately be called a normal physiological decalcification. There are cases, however, in which the degree of osteoporosis becomes so intense as to emerge into notice as a clinical disease. It is liable to occur extensively and persistently in certain stables. It is known to occur so often in this degree in connexion with certain types of dietary that it has been described as "bran disease." The condition disappears if the animal is sent away and put on good green pasture and it tends to recur after the animal returns to the stable. It has been described as affecting certain racing stables where the dietary was supposed to be of the very best.[24] Some veterinary practitioners have been unable to believe that they could be mocked at by a dietary containing so much of what they have always regarded as highly nutritious material and unimpeachable in quality. They have insisted that in the predilection for certain stables and in a tendency to enzootic prevalence at certain times or seasons there is proof that it is due to infection. But the preponderance of opinion is against this view.

It is equally difficult for some medical men to realize that in apparently liberal and even luxurious human dietaries the margin of safety in calcium or in certain amino-acid "building stones" (lysine, tryptophane, cystine) may be dangerously small and that deficiency therein might easily be a cryptic factor in chronic dyspepsia and other forms of ill-health. Unfortunately, no one can tell just where the line between sufficiency and deficiency lies and it might and probably does vary in the same individual as circumstances vary.

The calcium content of a dietary comprising meat, fish, eggs, soups, flour, bread, rice and other cereal foods, potatoes, peas, dried beans, sugar, butter and fruit is extremely small. Milk is the natural complement, but no doubt precipitated bone powder or even chalk would do.

Paroxysmal Hæmoglobinuria in Man.

Paroxysmal hæmoglobinuria in man, as in horses, occurs after exposure to cold, but the clinical picture is different in certain respects. A careful and scholarly description was written by Wickham Legg[25] fifty years ago. Material most useful for my present purpose is a paper by Copeman published thirty-four years ago. I give herewith an abridgement of portion of Copeman's paper.[26]

Copeman recorded his observation of seven cases, comprising four adults and three children. There was a history of syphilis in three of the adults, in the fourth it was perhaps less certain. All three children were subjects of congenital syphilis.

Case I.—Aged forty-one years. In the winter of 1885-1886 he began to suffer from coldness of the extremities and occasional "dying" of the fingers and in the same winter suffered from what was supposed to be frostbite of the toes. He was well in the following summer, but next winter whenever exposed to cold suffered from coldness of the extremities and "dying" of the fingers, shivering, and headache followed by the discharge of porter-like urine. He complained also of pain or difficulty in swallowing and was told that he was becoming jaundiced. In each succeeding summer he enjoyed good health, in each winter the malady returned. He was admitted to St. Thomas's Hospital on January 3, 1888. There was no evidence of disease on physical examination. In the ward his fingers used to "die" after washing with cold water and he occasionally suffered from uneasiness after food and from nausea. He was kept in hospital with his sanction for experiments.

Case II.—The patient worked in a foundry exposed to alternations of great heat and chills. For five or six years the urine had been dark on the average once a week. He was worse in cold weather. There were no physical abnormalities.

Case III.—Very similar to Case I.

Cases IV., V. and VI.—These were young children at Great Ormond Street Hospital. They were admitted for shivering fits in the winter of 1889-1890, the attacks being followed by discharge of blood-stained urine. They were weakly and half-starved when admitted. All improved greatly in health in hospital and for the most part the even temperature of the ward and good feeding prevented attacks. Two or three days' prevalence of specially cold weather was sufficient in each case to induce an attack.

Case VII.—A woman. She suffered from intense pain followed by hæmoglobinuria whenever the weather was very cold. There were no attacks in summer.

The following is a condensed record of the experiments on Case I.:

Experiment I.—Went out for a three-quarter-hour walk between 10 and 11 a.m. On return was cold and bluish and shivered for about an hour even though he sat before a warm fire. Hands also became blue and swollen, particularly three fingers of left hand. Throat also felt swollen and he experienced a pricking sensation on tip of tongue. Two hours after returning from his walk he passed a quantity of dark porter-coloured urine which deposited a dark sediment. The sediment consisted of granular particles, some of which were bound together into tube-casts. There were a few epithelial cells, but no blood corpuscles.

Experiment II.—Walked from 9.30 to 10 a.m., but did not seem to suffer inconvenience till his return. His temperature rose to 37.8° C. (100° F.) at 2 p.m. and he passed a quantity of dark urine. The blood examined immediately after exposure contained irregular coloured granules, the red corpuscles were irregular in shape and did not form rouleaux. At 6.30 p.m. the red corpuscles were found to be only 2,760,000 per cubic millimetre. They showed considerable variety in size and shape, some being twice as large as others. Here and there a slight attempt at formation of rouleaux was seen.

Experiment III.—The red cells numbered 3,665,000. Went out from 11.30 to 1.30, at which time his temperature was 35.6° C. (96° F.), it having been normal at 8 a.m. At 3.30 it had reached 39.4° C. (102.8° F.), after which it gradually fell to normal at 7 p.m. At 4.30 the blood was again examined, he having passed a quantity of very dark urine in the interval. The red cells now num-

bered 2,970,000. There was no attempt at rouleaux formation and the majority of corpuscles no longer retained their circular form.

Experiments IV. and V. were similar to Experiment III.

Experiment VI.—At noon placed his hands in iced water for ten minutes. The hands became white, "died," and he had a slight rigor. At 12.30 they were mottled white and red and still felt cold to the touch. The temperature before experiment was 36.6° C. (97.8° F.), at 1 p.m. it was 36° C. (96.8° F.), from 3 to 3.30 p.m it lay between 37.8° C. and 38.4° C. (100° F. and 101° F.) and had become normal at 7 p.m. The blood before experiment gave a red cell count of 3,765,000 and a hæmoglobin content of 39%. A little after 12.30 the count was 3,636,000, the hæmoglobin percentage being unaltered. The urine at 3.30 p.m. was reddish brown and contained albumin and amorphous pigment and a few white corpuscles, but no red corpuscles. At 6 p.m. it was a clear straw colour, but gave a guaiacum reaction. At 9 p.m. it had ceased to give a guaiacum reaction.

Experiment VII.—In the evening of the same day as Experiment VI an elastic band was fastened round the base of one finger which was then immersed in iced water for fifteen minutes after which a drop of blood was removed from the finger and examined. There was no formation of rouleaux and nearly all the red cells had lost their normal shape. Here and there three or four appeared to have become fused together and some were almost completely colourless, while the surrounding plasma was deeply tinged with pink. The blood taken at the same time from one of his other fingers was in what for him was the normal condition. Copeman performed a similar experiment on himself, but it produced no change in the ligatured and cooled finger. He mentions that Ehrlich and Boas had both performed similar experiments, tending to show that the injurious action of cold was purely local.

Experiment VIII. was similar to Experiment VI.

Experiment IX. was made to determine the condition of the urine at short intervals. The patient was out from 12.45 to 2 p.m. on a comparatively warm day, the shade temperature being 11.4° C. (52.5° F.). On his return his temperature rose only very slightly from normal. The urine at no time presented any trace of blood-colouring matter, but in place of it contained a small quantity of albumin which, however, Copeman found to be really globulin. The red cell count before he went out was 3,710,000, with a hæmoglobin value of 45% and on his return was 3,440,000, with a hæmoglobin value of 42%. The red cells formed poor rouleaux and were of various sizes, but well-shaped, before his walk. After his return they presented abnormal shapes and there was obvious escape of hæmoglobin into the plasma.

Experiment X.—At 4.30 p.m. the patient placed his hands in water at 4.4° C. (40° F.) for a quarter of an hour. Hæmoglobin appeared in the urine half an hour after removal of his hands from the water. No trace of methæmoglobin was detected, but after a specimen of the urine had been placed in the incubator at body temperature for two hours the hæmoglobin had become converted into methæmoglobin.

Experiment XI.—At 10.30 a.m. the patient had a cold bath in water at 14° C. (57° F.) in which he remained ten minutes. After the bath he had one or two slight rigors and complained of "pins and needles" in his hands which were white and cold. At 11 a.m. his temperature was 34.3° C. (93.8° F.) and by 12 it had risen only to 35.6° C. (96° F.). By 2 p.m. it had reached 38.2° C. (100.6° F.), after which it gradually fell till at 4 p.m. it was normal. The urine passed ten minutes after the bath contained neither albumin nor blood pigment. At 11.20 it contained hæmoglobin in large quantity. A specimen passed at 3 p.m. contained a small quantity of methæmoglobin.

There were some other experiments on other patients, but detailed accounts were not given as they were in accord with those described.

Copeman made the following remarks, among others:

"Dr. George Johnson, Dr. Mahomed, Dr. Ralfe and others have called attention to the fact that temporary albuminuria may follow cold bathing or other form of exposure to cold in otherwise healthy persons, while I have seen an attack of hæmoglobinuria follow the indulgence in a cold bath after extreme exertion at tennis in an athletic man in perfect health. . . . Dr. Ralfe last year wrote in *The Lancet* on paroxysmal albuminuria. . . . He expressly states that in four of his cases the attacks of paroxysmal albuminuria occurred in persons who had been subject to hæmoglobinuria."

Comments on Copeman's Material.

In considering the material presented by Copeman, the following features are worth particular note.

(*a*) Certain individuals show a predisposition, especially certain persons who have had syphilis.

(*b*) Exposure to cold causes an exaggerated and prolonged algide reaction. Coldness of the skin especially coldness and "dying" of the extremities is easily induced and the second phase of reaction is abnormally delayed. I would remark that during exposure to cold an algide reaction is protective and heat is conserved by it. The temperatures registered were probably from the mouth and do not necessarily indicate the internal conditions, but so far as superficial appearances go, heat production may at first have been low and over-balanced by heat loss. But notwithstanding the initial fall, the muscles must have afterwards got to work and after the cessation of the first phase there was a super-normal rise of temperature. Here heat production over-balanced heat loss. While the blood disintegration was practically immediate, as is shown by the finger experiment. there was a delay in the appearance of hæmoglobinuria. It may be reasonably conjectured that this was due to a paralysis of renal function during the algide phase.

(*c*) There is no evidence of degeneration of any muscle such as occurs in the typical azoturia of horses.

(*d*) No sugar appears to have been discovered in the urine. This contrasts with the azoturia of horses, if the French observers are right.

(*e*) There is definite proof that in a predisposed person the exposure of a finger to cold after applying an elastic band quickly produces disintegration of the red corpuscles locally and apparently nowhere else. Similar treatment in a healthy control individual failed to produce the phenomenon. There were very evident signs of physical alteration in red cells that were not actually disintegrated, namely swelling and irregularity of shape and loss of colouring matter.

(*f*) In Experiment IX. albuminuria was produced without evidence of hæmoglobin or methæmoglobin in the urine.

(*g*) Notwithstanding that usually a predisposition is noted, it is recorded that an attack has occurred in an apparently normal person after tennis and a cold bath. Likewise, transient albuminuria without hæmoglobinuria sometimes occurs and it appears that in some of these cases there has been a previous history of hæmoglobinuria.

While there is evidently much resemblance, notes (*c*), (*d*) and (*e*) point to some remarkable differences of form between the equine and human types

of paroxysmal hæmoglobinuria. The hæmoglobinuria symptom is common to both and in both there is a special tendency to its appearance in connexion with exposure to cold. In both it seems probable that there is an injury to the blood at some special part of the circulation, not everywhere, though afterwards the injured blood mixes with the uninjured. There is proof that in the human type the hæmolysis can be brought about by refrigeration and it would seem reasonably probable that the hæmolysis does actually take place in the parts that are reduced in temperature, and this could be made to explain the whole of the hæmoglobinuria. I am anticipating, but I shall later on refer to the "hæmolysin" theory of paroxysmal hæmoglobinuria in man, when it will be seen that the theory supposes hæmolysis to occur, not in the refrigerated area and during refrigeration, but at a later stage, when the blood reaches the internal organs. The finger experiment proves conclusively that it occurs in the refrigerated area and during refrigeration. Whether it also occurs in the internal organs the evidence from Copeman's cases enables us neither to deny nor to affirm. But it would seem to make it unnecessary. We are without information, but there is no evidence that hæmolysis could not also be produced in some animals by local refrigeration. It must be borne in mind, however, that in the equine disease the agency of chill appears sometimes to be absent.

It seems to me that a short supply of catalase need not be looked for as a factor in the hæmolysis that occurs in a refrigerated finger. Even in normal conditions the finger is not a place where intense oxidation is the rule, but so long as it is alive it requires oxygen to carry on its internal respiration and metabolism. However, when there is a serious fall of temperature in the part and still more when it is ligatured, the vital processes must diminish greatly. It is not dead, but its animation is more or less suspended. Whether it be also immersed in cold water or not, when a finger is ligatured both oxygen and catalase are cut off. Furthermore, when it is ligatured its supply of heat is cut off and its temperature will fall until it becomes approximated to that of the surrounding atmosphere. The mere fall in temperature is enough to interfere with oxidation, without considering catalase. In so far as oxidation is diminished, katabolism is rendered incomplete and there would be some tendency to accumulation of incompletely katabolized metabolites, that is of acidic substances.

This, however, would apply to every individual, predisposed or otherwise. The predisposition itself must be explained and then the mechanism of the condition might be cleared up. It is conceivable that if in certain persons the soluble calcium in the blood could somehow be diminished by refrigeration, an hæmolysis would occur as a consequence of a widening of the ratio between sodium chloride and soluble calcium—a disarrangement of ions. The normal ratio is 100 : 1 in gram molecules and if this were to be widened, an hæmolysis would be brought about. This, however, is pure speculation. There is no evidence. Furthermore, there is, as we shall see, another hypothesis.

I think I can say, after considering the evidence, that no data can be obtained from observation of the human material that will conveniently serve to explain the equine autolysis of muscles and selectively those muscles which have the most strenuous work, in which metabolism is most active and in which the greatest output of heat would naturally occur. We cannot postulate refrigeration there.

The same may be said about glycosuria. Apparently it is not found in the paroxysmal hæmoglobinuria of man. Yet it seems to occur at least sometimes in the equine disease and according to the French authors cited by Law it occurs in appreciable quantity.

The Hæmolysin Theory.

Up to this point I have abstained from mentioning an important theory that really occupies the foreground in regard to the paroxysmal hæmoglobinuria of man. I have done this designedly, for it seemed better to deal first with the equine disease and it enabled me to be able to present my own speculations with greater freedom. The theory referred to is at the present time the one generally favoured by authorities and for a description I abstract the following from Osler's well-known text-book.

> Much has been done latterly to clear up the nature of this remarkable disease by the studies of Eason, Donath, Landsteiner, Hoover and Stone and Moss. The blood serum of these patients contains a complex hæmolysin capable of dissolving the patient's own corpuscles. It is an amboceptor component of the hæmolysin, not the complement that is peculiar and this amboceptor differs from other known hæmolytic amboceptors in that it will unite with the red blood corpuscles only at a low temperature in the presence of a complement and furthermore in that it is capable of bringing about the solution of the patient's own cells (auto-hæmolytic action) and those of other members of the group to which the patient belongs as well as the cells of members of other groups (Moss). Atmospheric cold and congestion of the peripheral vessels, as in Reynaud's disease, will reduce the temperature of the blood sufficiently to permit of th eunion of the amboceptor and corpuscles and hæmolysis occurs when the blood passes to the internal organs.

The above theory appears to be regarded as satisfactorily accounting for the facts and therefore, provisionally, as proved. But we encounter a crucial difficulty in deciding what we shall include in the facts that have to be accounted for. It would seem satisfactory if we are on safe ground in assuming that there are two essentially distinct diseases, one commonly affecting horses, the other commonly affecting man. But as regards man, there would have to be excluded a group of cases, presently to be considered, probably classifiable with the equine disease.

For if the hæmolysin theory, as we may call it, is the true explanation of the ordinary human type, it seems to me that we have to admit at once that the equine disease is not homologous with the human. But are we entitled to say that the equine type is not homologous because it cannot be explained by a theory specially constructed to explain the

human type? That is like arguing in a circle. We choose the facts that are to be explained,and we find a theory that seems to be right because it fits the facts within the ring we have described around them. As far as I can see, the hæmolysin theory is not able to explain the muscular degeneration that occurs in the equine disease, nor is it able to explain the glycosuria that has been recorded. It may be said that the equine disease is "obviously" different and if all agree in that, it will stand as a convention. This is done every day in classification and we are usually justified by commonsense in doing it. But if someone objects that by doing so we are merely shutting our eyes to a real difficulty and effectually begging the question, it can be no answer to use the circular argument that the theory demands it. That is not the way to test theories. We begin to suspect them when we find that they only fit a portion of the facts within the natural area. On the other hand, if the two types are essentially different we do not need to have a theory coordinating both. But that is a question at issue and there is our dilemma.

But let me not be misunderstood. The foregoing remarks apply quite as much to my theory of the causation of the equine type of paroxysmal hæmoglobinuria as they do to the hæmolysin theory in connexion with the human type. I confess to a degree of dissatisfaction in not finding coordinating factors and a coordinating theory. It is possible that some day someone may visualize a coordinating theory. On the other hand, this may really be impossible because the differences are essential.

However, hypotheses are servants, not masters; there is nothing sacrosanct about them. They are merely provisional explanations of the facts as far as we know them at present—and there is much more to learn.

Hæmoglobinuria from Exertion: Is the Equine Type of Hæmoglobinuria Represented in Man?

The hæmolysin theory is described in detail by Eason.[27, 28, 29] At the conclusion of an article on hæmoglobinuria in the second edition of the *Encyclopædia Medica*, Eason refers under a separate heading entitled "Hæmoglobinuria from Exertion" to a class of cases of paroxysmal hæmoglobinuria of comparatively rare occurrence that have been recorded as following exertion and independent of cold. He remarks on the curious feature that in these cases it is apparently leg work that induces these attacks. Admitting that symptoms of acute muscle degeneration are not in the description, nevertheless the similarity to the equine disease is too close to miss.

It is obvious that these cases are not amenable to the explanation given by the hæmolysin theory as set out, wherein refrigeration is an integral necessity, a fundamental factor. This is a practical example of the dilemma to which I have referred in a recent paragraph. Either the theory in its present form breaks down or the disease is a different one, despite its superficial resemblances. If we take the latter alternative, it seems reasonable to class it with the equine type. If my hypothesis as to the causation of the latter type is near the truth, it might then be found to explain these human cases.

We have seen that in slight attacks, both of the equine and of the human type of the disease, there may be no appearance of hæmoglobin in the urine, but albumin only. This causes us to reflect. Let me now quote from an article by Dr. W. Collier, of Oxford,[30] on functional albuminuria in athletes. He shows that after violent exercise in young men it is practically universal to find albuminuria:

> Of this year's Oxford University crew every member after rowing a trial over the full course passed a definite amount of albumin and at least half the crew passed a very considerable quantity. With the college crews the same thing happened. In the New College boat, head of the river in the Torpids, after rowing a course every member's urine contained some, while in five of the crew the amount was large. The running men seemed to pass even more than the rowing men.

We may now recall to mind the case of hæmoglobinuria observed by Copeman in a healthy man after tennis and a bath.

Can it be that the hæmoglobinuria that occurs in some persons after exercise, meaning the kind that I have classed provisionally with the equine type, is a sort of pathological superlative, an exaggeration of a normal occurrence that in ordinary people reaches no higher than an albuminuria?

Summary.

The ætiology and pathology of paroxysmal hæmoglobinuria, equine and human, is discussed. In the horse, reasons are given for believing that the dietary is liable to be very low in calcium through the nature of the customary dietary and the enormous variations that occur in the calcium content of oat hay or straw. For example, a sample of oat straw grown in one soil or region may contain six times as much calcium as a sample grown in another soil or region and lucerne hay may contain fifty times as much. Actual figures are quoted. The physiological regulation of calcium metabolism may also be at fault.

It is thought likely that in the horse functional liver disorder promoted by calcium deficiency and/or by chill may lead to deficient catalase production. The acute degeneration occurring specially in the great thrusting muscles of progression in the horse at the same time as the paroxysm of hæmoglobinuria is explained as arising from exhaustion of catalase, with consequent deficient oxidation and accumulation of lactic acid. Proteolytic enzymes are at the same time set free and their activity, previously inhibited, is no longer restrained. Autolysis is the result and hæmolysis accompanies it.

The glycosuria that occurs in the equine, but not in the human cases is accounted for by inability to oxidize sugar in the affected muscles, although it has been demanded and supplied. Therefore it accumulates and has to be got rid of.

In the typical paroxysmal hæmoglobinuria of man, evidence is cited showing conclusively that hæmolysis occurs within the refrigerated part, in the cold blood then and there present. The evidence at hand fails to support the now current view that

the hæmolysis occurs in the internal organs at a period subsequent to the chilling. It does not contradict it, but it makes it superfluous.

The hæmolysin theory, at present the prevailing interpretation of the paroxysmal hæmoglobinuria of man, is inapplicable to explain the equine disease. It has not been found possible to coordinate and connect together the equine type with the type described as most frequent in man. If both have been interpreted correctly, they are not homologous, but essentially different diseases, despite resemblances. It is noted, however, that in man two distinct types of paroxysmal hæmoglobinuria appear to occur. It is suggested that the second and less common of these types may be homologous with the equine disease.

Having regard to the fact that albuminuria occurs normally in human beings during strenuous exercise, the question is raised whether the equine type of paroxysmal hæmoglobinuria is not the homologue of this, though extended into a pathological plane.

Incidentally, it is suggested that the phenomenon known as "second wind" in athletes is due to establishment of catalase equilibrium in response to a demand for a greater output from the liver.

It is also suggested that the pain and stiffness that occurs in an over-used untrained muscle may possibly be due to a muscular change similar in kind to that which occurs in horses suffering from paroxysmal hæmoglobinuria, but only very slight in degree.

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SULPHURETTED HYDROGEN POISONING: WITH SPECIAL REFERENCES TO EYE SYMPTOMS—"PINK EYE."

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SULPHURETTED hydrogen is produced in Nature by the putrefactive decomposition of various animal and vegetable substances containing sulphur and is found sometimes in mine air.

Gases in the air act either (i.) by exerting a poisonous action on the animal system or (ii.) from

deficiency in oxygen caused by dilution of air with gas, the gas itself having no specific action.

Sulphuretted hydrogen comes under the first heading. It is a powerful poison which when inhaled acts through the blood stream, producing headache, smarting of the eyes, confusion of mind and loss of power over limbs. The individual developes a drunken gait and may even pass on to a stage of insensibility followed by death from asphyxia.

Eye Symptoms.

The eyes feel as though full of sand and are streaming with water. The patient's eyes can only be opened in a darkened room. There is intense photophobia, men often having to be led home. The local signs are acute lachrymation, with conjunctival congestion and œdema accompanied by blepharospasm. Acute symptoms last about two to three days.

It is considered that the chief action of the gas is through the blood stream causing irritation to mucous surfaces. This is borne out by the facts noted clinically. In the first place patients have frequently been observed to develope ocular symptoms twelve to twenty-four hours after leaving their work, having had no local symptoms while exposed to the action of the gas in the mine. Secondly, affected patients suffer from inflammatory reaction of the intestinal tract and this often leads to acute diarrhœa. It is questionable whether the gas has any direct action upon the conjunctiva, unless in very concentrated form. In Haldane's book, "Investigation of Mine Air," he quotes Lehmann who asserts that sulphuretted hydrogen acts locally as well as indirectly after absorption into the blood stream. This assertion is not borne out clinically in my experience, except under the conditions already mentioned.

Poisonous Properties of the Gas.

According to Lehmann 0.05% to 0.07% produces serious symptoms if not death in man after exposure for about one hour. Haldane states that in his experience the action of sulphuretted hydrogen is very sudden and death often occurs in men who have received a sufficient quantity of the poison, even after they have been brought up into the air. It is considered the most poisonous gas found in coal mines (Le Neve Foster and Haldane's book, "Investigation of Mine Air").

Detection.

The odour of the gas is often a useful indication, but Haldane considers this unreliable. He states that he has detected sulphuretted hydrogen in pit air with lead paper when it was impossible to detect any odour.

Treatment.

Palliative measures have been tried by using castor oil drops, but without success. Personally I am against local palliative measures because of the possibility of masking symptoms and getting a fatal dose of poisoning. The gas seems only to cause the distressing symptoms if it is very concentrated. If sufficient air can be got through the affected area the men are able to proceed with the work.

A place which was very bad in one of the local mines improved as soon as a "cut through" was made into another drive, allowing the current of air to circulate through direct from the main shaft. The indications therefore point towards attempts being made to cause a better circulation of air through affected area. Intermittent work may be allowed at this area after local testing with lead paper. In affected patients twenty-four to forty-eight hours out of the pit is generally all that is needed. Local soothing drops such as argyrol or tincture of opium and distilled water in equal parts may be used.

It has been noted as a matter of experience that each attack of "pink eye" renders the individual more susceptible to further attacks. Lehmann states that except in great concentration sulphuretted hydrogen causes no characteristic change in the blood.

Reports of Cases.

UNILATERAL FUSED KIDNEY.

By ARTHUR A. PALMER, M.B., Ch.M. (Edin.), F.R.C.S. (Edin.),
Government Medical Officer, Sydney.

IN THE MEDICAL JOURNAL OF AUSTRALIA of March 15, 1924, W. B. Bieberbach is quoted as reporting an instance of unilateral fused kidney. He stated that about seventy instances of this condition have been reported. No doubt many other instances have been seen on the *post mortem* table, but not reported.

In Bieberbach's patient the condition was diagnosed during life. The following instance was seen on the *post mortem* table.

A man, aged forty-six years, was brought to the Sydney Morgue dead from a fractured skull. The left kidney was absent. The right kidney was large and its two ureters passed downwards ventral to the renal vessels. Near the brim of the pelvis the lower ureter crossed to the left and finally entered the bladder as the left ureter, the other one continuing on as the right ureter. Both suprarenal capsules were present and in their usual situations. The specimen was, I think, sent to the Sydney University Museum.

A CASE OF SARCOMA OF THE ISCHIUM.

By R. M. CROOKSTON, M.B., B.S. (Melbourne),
Camden.

I VENTURE to report the following case which ended four years ago, because of the many unusual features which it presented.

Mrs. B.H., *ætatis* twenty-one years, consulted me in 1917 because of a vague and indefinite pain, sometimes severe and spasmodic in character, radiating down the front of the left thigh. Pain had been present since her confinement four months previously and she stated that labour had been prolonged (two days) and terminated by forceps. Examination on this occasion revealed nothing, except for a soft indefinite thickening in the left lateral fornix and some tenderness on pressure over the front of the left thigh below Poupart's ligament.

A few weeks later she discharged *per vaginam* about a litre of clear fluid. Shortly afterwards I opened the abdo-

men and removed the left tube which had obviously been recently dilated, though at the time of operation it was collapsed. The condition had apparently been hydrosalpinx and a good prognosis was given for her future. After a short interval, however, she began to complain of pain referred to the distribution of the left sciatic nerve and for about six months the pain was troublesome with intervals of freedom. There were moderately frequent occasions when the pain commenced usually at night with great violence. The type of pain was neuralgic and was severe enough to necessitate the administration of morphine.

After about six months' intermittent duration she became almost completely free from sciatic pain and appeared in good health for two months. She was then seized suddenly with violent abdominal pain. Within twelve hours of the onset of pain signs of intra-peritoneal hæmorrhage were present. I re-opened the abdomen and removed the right ovary with a ruptured gestation sac attached. Her periods had been irregular during my whole attendance and the diagnosis of the condition was difficult until the signs of hæmorrhage occurred. The condition was apparently a true ovarian pregnancy ruptured probably during the first month of gestation. Recovery was uneventful and she again appeared to be in good health for a few months. At the end of this time she consulted me on account of a painless mass in the left thigh immediately below the middle of Poupart's ligament. Exploration with a syringe resulted in a ten cubic centimetres barrel full of blood and an X-ray photograph by the late Dr. Herschel Harris revealed the presence of a sarcoma of the ischium. From this time on the progress of the growth was rapid and obvious. Within two months a mass presented in the abdomen on the left side, smooth, firm and rounded, reaching as high as the umbilicus. Injections of "Cuprase" were given and during the eight weeks occupied by a course of injections, the growth shrank rapidly till it was just palpable above Poupart's ligament. The patient then refused injections on account of the great pain they caused and within three months the growth reached nearly to the level of the ribs, apparently filling about four-fifths of the abdomen. It also caused huge deformity of the buttock.

She continued to go about and attend to much of her housework for some weeks and then retention of urine occurred. From this stage the end came quickly. She had up till the day retention occurred been plump and apart from the deformity of the growth, a well-looking girl. Within a week she had wasted to a wreck of suffering humanity and died.

I submit the history of this case without comment.

Reviews.

RESPIRATORY DISEASES.

Two well-known French physicians, Professor F. Bezançon and Dr. S. I. de Jong, have collaborated to produce a text-book on diseases of the respiratory organs.[1]

The work is condensed, but not to such a degree as to deprive it of those personal touches which lend interest. In this respect it recalls the late William Osler's "Medicine," as also in the occasional historical and philosophical digressions which add to its literary charm. These are specially striking in the articles on pneumonia and on tuberculosis.

It opens with an essay on the parts played in respiratory diseases by infection, by mechanical difficulties, by failures of circulation and by nervous disorders. The authors deal shortly with the anatomy and physiology, normal and morbid, of the lungs and then proceed to a methodical study of the various portions of the respiratory system from the nose downwards. The discussion is limited to medical affections. Pneumonia is considered in a number of aspects, for example as a pneumococcal alveolitis and as a pulmonary pneumococcosis. Laennec's description of the gross morbid anatomy is not only followed, but actually quoted *verbatim*, page after page. This tribute to the master is well deserved, but surely it is extreme to refrain from mention of the rib markings on the exterior of the hepatized lung because he denied their occurrence.

Our authors have broken away from French tradition by including acute congestion of the lungs—described separately and at great length by Dieulafoy as *maladie de Woillez*—under pneumonia, lobar or lobular. The article on pulmonary tuberculosis covers one hundred and ten pages. It contains outlines of the history of the various conceptions that have obtained from the time of Hippocrates onwards, and includes a description of the "modern conception" of the process in a series of propositions of intense interest. These are already so condensed that it is only with hesitation that we essay to summarize further as follows: (i.) Pulmonary tuberculosis almost always arises by inhalation; (ii.) the primary lesion is in the lung itself constituting an inoculation chancre, defined and symptomless; (iii.) from this in accord with Parrot's law the bronchial glands are infected and a more important lesion results; (iv.) in the infant the primary chancre and the adenopathy are often followed by a rapidly mortal generalization; but (v.) if the bacilli are few or the resistance is high the disease is manifested as a tracheo-bronchial adenopathy and the glandular lesions constitute "a veritable reservoir of virus," sometimes encysted and latent; (vi.) from this reservoir the germs again reach the lung by irruption into a bronchus (bronchial embolus) or into a pulmonary vein; (vii.) the lesions of this second pulmonary invasion may be active and manifest or latent; (viii.) tuberculosis appearing in an urban adult is rarely a recent infection: it is the re-awakening of a latent infantile infection; nevertheless we speak of it as early pulmonary tuberculosis.

The adoption of this "modern conception," so distinct from that of their illustrious compatriot Calmette, constrains our authors to treat tuberculosis on a scheme quite different from that suitable for other infective diseases. They do this in a series of chapters which contain all that would be expected, though in an unusual arrangement. Rare clinical forms, such as the emphysematous—and rare forms of phthisis are commoner than many well-known diseases —are duly noticed; so too is the differentiation of descending rhino-bronchitis. Where so much is excellent, we may be excused a regret that the value of tuberculin in diagnosis is so cavalierly dismissed and that Bezançon and de Jong know so little of recent therapeutic developments as to use almost exclusively Koch's old tuberculin.

The book claims to be merely a *précis* and in places the information given is scant; for example treatment is sometimes very briefly described and little is said of the late effects of poison gas of war, except that it usually leads to fibrosis and rarely to tuberculosis. Can our French comrades have been so much more fortunate than our own men?

Elsewhere room has been found for much recent and interesting work; the account of physical signs in pleurisy with effusion is specially remarkable. We learn not only of the familiar semi-lunar space of Traube and paravertebral triangle of Grocco, but also of the thread sign of Pitres. According to this sign a thread hung from the sternal notch of a patient with pleurisy deviates from the *symphysis pubis* towards the affected side so as to make an acute angle with the sternal axis. Mention is also made of the parabolic curve of Damoiseau along the upper limit of the effusion and of the resonant triangle of Garland between this curve and the spine. As is usual in French text-books there is no index. Of the many authorities quoted nearly all are French; no references to their writings are given.

The volume is one of a series to other members of which the reader is referred for some information. The binding, paper, printing and illustrations are excellent. We cordially recommend it.

[1] "Précis de Pathologie Médicale: Tome II., Maladies de l'Appareil respiratoire, par MM. le Professeur F. Bezançon et le Docteur S. I. de Jong; 1923. Paris: Masson et Cie; Post 8vo., pp. 566, with 83 figures. Price: Frs. 25 net.

SEXUAL DISABILITIES.

CASES of impotency and barrenness are met with from time to time and Dr. Frank P. Davis's book on impotency, sterility and artificial impregnation is designed to assist the practitioner in dealing with them.[1] That a second edition has been called for speaks in its favour.

The author attaches great importance to the bearing which the senses of smell, hearing, sight and touch have upon sexual excitation and sexual power, and discusses the production or inhibition of excitation by various perfumes, body odours, tone and inflection of voice, music and certain characters of the female form. This section of the work is gone into in detail and numerous examples are given from case records and other sources.

While there is much to be said for these arguments regarding the production of sexual excitation, there is no doubt a limit to the part played by the special senses and the author seems to have claimed for them at least a full measure of importance. It would appear that something more than the sense of hearing plays a large part in the production of the orgasms which one lady could experience while listening to music and the opinion that there would be very few separations and divorces "if people only mated with those whose voices were in tune with themselves" is a courageous one.

The causes of impotency are classed as surgical and psychical and prominent among these are various anxiety states and mental preoccupation. In describing methods of treatment emphasis is laid on the importance of suggestion —the Decameron is hinted at for the girls who are indifferent because they cannot keep their minds away from the stories they have been reading!

With regard to sterility various surgical, mechanical and postural methods of treatment, designed to ensure the germ and sperm cells in the proper environment are described. It is in this connexion that the indications for artificial impregnation and the methods applicable are discussed.

Cases in which the desire for children remains unsatisfied are by no means rare, nor do they readily respond to treatment. As the author says, the day when these patients could be given a tonic and told to forget their troubles, is past.

The subject is of interest to the practitioner and the book holds out suggestions which he should find of value in investigating the anomalies with which it deals.

THE CHEMISTRY OF TUBERCULOSIS.

"THE CHEMISTRY OF TUBERCULOSIS" is the title of a recent book by Wells, De Witt and Long, which should prove to be a most useful work of reference to all who are interested in tuberculosis.[2] It provides an excellent critical review of the more recent work on this subject together with a comprehensive bibliography.

In the first section of the book Dr. Long deals with the chemical composition of the tubercle bacillus and its products, furnishes some interesting data concerning the metabolism of the organism and discusses in the light of present knowledge the phenomenon of "acid-fastness."

Professor H. G. Wells contributes the second portion of the work which is concerned with the chemical changes which result from infection. Of necessity it covers a very wide field, comprising the study of tuberculous and non-tuberculous tissues in the infected host and the chemistry of the blood, urine and sputum in tuberculosis. Of particular interest is the consideration of the metabolism of the tuberculous subject. In non-febrile patients there appears to be but little change in the basal metabolic rate, but in patients with fever the increased heat production is chiefly at the expense of the carbo-hydrates, fat combustion occurring from chills and increased muscular activity, while the destruction of tissue protein results from intoxication and not from the fever by itself. The daily excretion of nitrogen in patients taking diets containing little protein and much carbo-hydrate is about twice that of the healthy subject, but a nitrogen balance can be obtained in most cases by the administration of sixty to seventy grammes of protein *per diem*. Since the specific dynamic action of protein leads to increased pulmonary work, Wells concludes that diets in which protein is largely in excess of the minimal requirements, are not desirable in actively progressing febrile cases. The study of mineral metabolism shows that "demineralization" does not occur in the tuberculous.

The last part of the book is an admirable critical survey of the chemo-therapy of the disease. Dr. Lydia De Witt has a fascinating, if up to the present unproductive, theme. In discussing the experimental work which has been performed chiefly upon guinea-pigs which are much more susceptible to tuberculosis than is the human subject, she points out that though no drug has yet cured the disease in these animals, in some cases at all events the lesions have been rendered less progressive and less widely spread than in control animals. A rational basis for treatment involves a knowledge of what any particular drug does in the normal body and "a reason for the belief that it acts beneficially in a specific manner on the tubercle" either directly by killing the bacillus, checking its growth or neutralizing its products or indirectly by increasing or diminishing phagocytosis, increasing the destructive power of the phagocytes, by cutting off the oxygen supply and allowing accumulation of waste products to destroy the bacilli by means of the proliferation of surrounding tissues or by causing mineralization of these tissues and thus limiting further spread. In any case it is of fundamental importance that the drug used should permeate the lesions. This conception of chemo-therapy is a good deal wider than Erlich's original idea of the search for drugs which should possess a maximal effect on the parasite combined with a minimal action on the host, but even the action of "Salvarsan" is by no means so direct as was at first thought to be the case. The action of the dyes and their compounds with heavy metals, of creosote, guaiacol and their derivatives, of fats and the fatty acids, of the heavy metals, of iodine and of the rare earths are all considered in the search for a specific therapeutic agent which has so far been without success and until some such agent is found we must rely upon "dietetic and hygienic measures as the main hope in the treatment of tuberculosis."

ACUTE ABDOMINAL DISEASES.

THE second edition of Adams's book on the diagnosis and treatment of acute abdominal diseases has been received.[1] The book is now written by Joseph E. Adams alone, as his collaborator in the first edition is devoting his time to other work.

In this edition which is well produced, extensive in scope, simple in language, the author deals with the subject in a very able manner. Some criticism could, of course, be levelled at it. We might complain that in dealing with femoral hernia, no mention is made of Dujarier's operation. Again, the statement that gastric peristalsis causes no pain may not be conceded by those who regard the pain of gastric ulcer as being due, in great part at least, to muscular action. The perusal of the article on gastric and duodenal ulcer gives the impression of something unfulfilled, something just missed. But such criticism would be strained as the book is a very able discourse on a most important subject. We can thoroughly recommend the work to students and practitioners and even the specialist may learn something from it.

[1] "Impotency, Sterility and Artificial Impregnation," by Frank P. Davis, Ph.B., M.D.; Second Edition, Revised and Enlarged; 1923. St. Louis: C. V. Mosby Company; Post 8vo., pp. 168. Price: $2.25.

[2] "The Chemistry of Tuberculosis," by H. Gideon Wells, M.D., Ph.D., Lydia M. De Witt, M.D., A.M., and Esmond R. Long, Ph.D.; 1923. Baltimore: Williams & Wilkins Company; Demy 8vo., pp. vii. + 438 and index. Price: $5.50.

[1] "Diagnosis and Treatment of Acute Abdominal Diseases Including Abdominal Injuries and the Complications of External Hernia," by Joseph E. Adams, M.B., M.S. (London), F.R.C.S. (England); Second Edition; 1923. London: Baillière, Tindall and Cox; Demy 8vo., pp. 568, with forty-six illustrations. Price: 16s. net.

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The Prognosis of Syphilis.

IT is probably correct to say that in no disease is prognosis a more difficult problem than in syphilis. From whatever aspect the subject is approached grave difficulties are encountered. In its early clinical manifestations syphilis is an obvious though not lethal affliction. In its later developments and more remote sequelæ it is less obvious and makes its deadly presence felt in a more insidious and subtle manner. Although *tabes dorsalis* and general paralysis of the insane are common causes of death, the word syphilis does not appear on death certificates. It is nevertheless widely disseminated and appears dressed in the more respectable and less compromising garb of aortic aneurysm and vascular changes and makes its malign influence felt in certain forms of mental disease, paralysis, nephritis and so on.

In considering the question of prognosis the first thing to be remembered is that with modern methods of treatment, undertaken at an early stage and pursued with energy, syphilis is a curable disease. This has been proved by the record of undoubted second infections recorded in the literature in persons who have been treated by arsenic and its derivatives. With the old methods of treatment it is doubtful whether second infections ever occurred and hence whether cure was ever obtained. The every day application of the Wassermann test to the treatment of syphilis has been of immense assistance in indicating the progress made by the patient in his response to therapeutic measures. Great caution, however, must be exercised in relying on its results in prognosis. The continued persistence of the power to react on the part of the serum or cerebro-spinal fluid of a patient must be taken as an indication of the presence of spirochætes in his tissues and of the necessity for further treatment. It is well known, too, that a reaction may be obtained with the blood and not with the cerebro-spinal fluid and *vice versa*. More than this, both the blood and cerebro-spinal fluid may fail to produce a reaction for a considerable length of time and the power to react may subsequently return in one or other medium. From this it will be seen that the only real criterion of cure lies in the impossible expedient of exposing an individual to the risk of a second infection. These facts should be remembered by a practitioner when confronted with the task of giving permission or refusing it to a syphilitic patient desiring to marry. When faced with such a problem the medical practitioner will only find real difficulty in regard to the question of infectivity. The question of the risk of the subsequent development of late syphilitic manifestations in the nervous system will probably be dismissed when the subject of marriage is being discussed.

In regard to infectivity it is obvious that the absence of a reaction to the Wassermann test with the blood serum or the cerebro-spinal fluid or both is no guarantee whatever that the patient will not infect his wife or beget children who will manifest signs of congenital syphilis in some form or other. It is a matter of common knowledge that a patient with signs of active syphilis may beget children who are apparently quite healthy and that stigmata of the disease may be borne by children whose parents manifest no signs of it. In these circumstances the question may well be put as to whether a syphilitic patient should ever be allowed to marry. There are several other considerations to which careful thought must be given. Most authorities are agreed that much depends on the way in which the patient has responded to treatment and on the length of time during which there have been no clinical manifestations of the disease. If a patient whose blood serum and cerebro-spinal fluid fail to yield a reaction in the Wassermann test, has had no clinical manifestations for some considerable time and if at the time of treatment he responded in a normal and regular manner to the various therapeutic measures, it is probably justifiable to allow that patient to marry. Each case must be judged on its own merits and no hard and fast rule can be laid down.

In discussing this subject it is necessary to emphasize and re-emphasize the fact that the inability to obtain a reaction in the blood serum or the cerebro-spinal fluid does not indicate an absence of infection. As a measure of the patient's immunity and an indication of his response to treatment it is invaluable. Moreover, the occurrence of a reaction always indicates the presence of spirochætes in the tissues of the patient. It were far better to ignore the failure to produce a reaction altogether, than to attribute to its absence indications which have no foundation in fact. It is well also to refer to a matter which is of importance and is the cause of much misapprehension in connexion with the Wassermann reaction. The use of the word negative in describing the absence of a reaction is misleading. It is a contradiction in terms. Absence of reaction is merely the *status quo ante*. By its use medical practitioners are unconsciously led to attribute to the result of the test something which may be unjustified. Clear thinking in regard to ascertained facts will undoubtedly lead to a fuller understanding of the hazardous nature of a prognosis in syphilis.

Current Comment.

SPRUE AND CŒLIAC DISEASE.

THE similarity of some forms of cœliac disease to the condition known as sprue has frequently given rise to the suggestion that the two may be merely different manifestations of the same pathological process. Samuel Gee who originally described cœliac disease in 1888, was the first to see the resemblance and associate the conditions. Others differed from him and refused to recognize the association on clinical grounds alone. Cheadle held that there was a condition which he described as acholia and was to be distinguished from cœliac disease proper. As a matter of fact the ætiology of both sprue and cœliac disease is by no means determined. In regard to cœliac disease a micro-organism and an error in metabolism have both been held responsible and the occurrence of sprue has been attributed to glandular insufficiency, yeasts, micro-organisms and so forth. Among the more prominent investigators into the causation of sprue has been Ashford, of Porto Rico. Reference was made to his work in THE MEDICAL JOURNAL OF AUSTRALIA on May 12, 1923. Ashford holds that two factors are responsible for the appearance of sprue. There is in his opinion a glandular insufficiency on which is grafted the appearance in the alimentary canal of the *Monilia psilosis*. He regards this fungus as the specific organism concerned in the production of sprue.

The ætiology of sprue and cœliac disease and their relationship to one another were recently the subject of discussion before the Royal Society of Medicine.[1] The remarks of some of the speakers were both interesting and important.

Sir Leonard Rogers, F.R.S., said that sprue was either produced by several different factors or else its essential cause was still undiscovered. First of all he alluded to the work of Ashford on *Monilia psilosis* and stated that patients' sera gave complement deviation tests with its cultures. Vaccines made from the monilia had proved of value in treatment. Ashford had also laid stress on the predisposition of glandular deficiency due to dietetic and climatic conditions. Sir Leonard Rogers referred to his own successful treatment of a considerable number of patients suffering from sprue in the tropics with autogenous oral streptococcus vaccines. He had thought that streptococci played an important part in the ætiology and Nichols had confirmed the value of this line of treatment and had identified the streptococcus as belonging to the viridans groups. The incriminated organism, however, was not constantly present and relapses might occur after the vaccine treatment. He thought the streptococcal infection was possibly of a secondary nature. In regard to digestive disturbances both Brown and Bovaird had reported a considerable deficiency or absence of pancreatic ferments. The chief result of this deficiency was seen in the excess of fats in the bowel mainly in the form of fatty acids instead of as neutral fats. This with the formation of the characteristic stools brought sprue into close relationship with cœliac disease. In typical advanced sprue it was impossible to decide the relative importance of the digestive and infective factors in the production of the disease. It was necessary to turn to the early stages of the disease. In India "hill diarrhœa" was a preceding condition in about 20% of patients. Hill diarrhœa was essentially a physiological digestive deficiency. All the other predisposing causes of sprue were of a debilitating nature and he thought that the onset of the disease was probably due to some digestive weakness which later on became aggravated by secondary infection. The rôle played by vitamins in the ætiology of sprue had been discussed and not without reason. Cases of sprue had been reported as occurring in patients in whose diet there was no deficiency of vitamins. He suggested, therefore, that in sprue there was a diminished absorption of these substances owing to intestinal atrophy and reduced intestinal ferments.

Dr. Reginald Miller discussed cœliac disease. He said that there were such notable differences between the two conditions as to make it unlikely that they were the same. He outlined the clinical picture of cœliac disease and said that its essence was a malabsorption of the fat in the diet. Cœliac

[1] *Proceedings of the Royal Society of Medicine*, February, 1924.

disease was not a form of intestinal disease in which diarrhœa became so severe that fatty stools were passed. The primary factor was a digestive fault and symptoms of enteritis were secondary. This was the important fundamental conception of the disease and was proved by the fact in mild cases no diarrhœa occurred, in severe cases there was no diarrhœa in quiescent intervals or during convalescence, but the stools on analysis showed a large excess of fat. There was also the fact that in *post mortem* examinations there was no evidence of enteritis or of intestinal atrophy. It was known that the disease was not due to a persistent inhibition of bile flow. Dr. Miller thought that it was not unlikely that it was due to some abnormality in bile salt secretion. In this connexion it is interesting to remember that Cheadle thought that the pallor of the stools was due to the complete absence of bile. Cammidge found slight deficiency of bile in the stools of some patients, but Still pointed out that the colour was in large measure due to the presence of undigested milk fat.

Dr. Castellani referred to the streptococcic theory of the origin of sprue and to the view as to its monilial origin. He expressed the opinion that streptococci and moniliæ only produced certain symptoms of the malady and were not causative agents. He also referred to the view that sprue was an old manifestation of dysentery, especially of amœbic dysentery. This was interesting as according to it sprue might be regarded as of amœbic origin. It would, therefore, in a certain way correspond to the tertiary stage of syphilis.

Dr. H. H. Scott described a method of treatment which he claimed possessed a rational basis. He had elaborated the method by studying the history of a patient whose symptoms included frequent cramps or carpo-pedal spasms. He had endeavoured to trace the symptoms to a common origin. The stools and urine had shown a relatively large excretion of calcium. He had thought it possible that the acidity, aphthæ, diarrhœa, loss of weight and languor might arise from intestinal intoxication. He next had thought of the association of carpo-pedal spasms with calcium deficiency, the possible correlation of these with the diarrhœa and the intestinal toxins and their reference to the para-thyreoid glands which were supposed to control both these conditions. Examination of the blood had revealed that the total calcium was little if at all diminished. He had, therefore, inferred that in spite of the loss of calcium in the excreta, sufficient was being absorbed to maintain the total calcium in the blood. Dr. Scott pointed out that calcium is present in the blood in two forms—the ionic or free and the combined or coagulative. He had found that the coagulation time was normal, so there was no deficiency of the latter. He had therefore inferred that the ratio between the two was possibly upset. Lactate of calcium had been administered to the patient and temporary improvement had resulted. Para-thyreoid extract had then been given and the patient had improved at once. The symptoms had disappeared and since that time the patient had lived a normal life. Dr. Scott then proceeded to review the symptoms associated with calcium deficiency and to apply them to sprue. He said that in countries in which sprue was endemic, the methods of cooking led to an increased ingestion of fat. Kochmann and Petzsch had shown that a considerable loss of calcium occurred by adding fats to the diet of healthy dogs and Rothberg and others had noticed that in children to whom an excess of fats was given, there was a reduced retention of calcium. Korenchevsky also had found that in animals on a diet deficient in calcium diarrhœa occurred together with such symptoms as loss of weight and increased nervous excitability. The mechanism of the development of this diarrhœa had not been clear to Korenchevsky and he had thought that it might be of importance in elucidating the problem of some forms of infantile diarrhœa. Dr. Scott said that it would be of interest in connecting sprue with cœliac disease. The amount of calcium in the blood depended on the efficiency of the calcium regulating mechanism. This mechanism consisted in the para-thyreoid glands with their two-fold function of de-toxication and regulation of calcium metabolism. Dr. Scott summed up his views by saying that in sprue one of two conditions obtained. In one where the fats were in excess, there was an excessive excretion of calcium in addition to intoxication of intestinal origin, in the other there was protein excess with intestinal toxin formation. In both the para-thyreoid detoxicating function was overburdened with a resultant disorganization of its calcium regulating function while there might be in addition a diminished calcium absorption. There was interference with both functions of these glands.

Dr. Arthur Powell drew attention to the absence of pathological changes at *post mortem* examinations of patients who had suffered from cœliac disease. He compared the course of the two conditions and said that it was obvious that they were two distinct entities. Vitamin deficiency was not a cause of sprue for it occurred more often in persons whose diet was not deficient.

Dr. P. Manson-Bahr said that he should very much like to know what was the essential lesion of sprue. In order to discover the essential lesion it was necessary to examine at autopsy patients who had died while in the early stages of the disease. He had had two opportunities of doing so. Both patients had been comparatively young men and in both the disease had been of short duration. In both the small gut had been extensively ulcerated and both had died from septic peritonitis following on perforation of an ulcer. He believed that an ulceration of the ileum was the essential pathological lesion and that the lesions were similar in extent and origin to those which occurred in the mouth.

Dr. J. A. Ryle on the basis of some clinical histories suggested that in both conditions there might be some obstruction of the mesenteric lymphatics, probably inflammatory. The calcium deficiency would be due to the excessive excretion of calcium in the forms of soaps.

Abstracts from Current Medical Literature.

GYNÆCOLOGY AND OBSTETRICS.

Innervation of the Uterus.

BECKWITH WHITEHOUSE AND HENRY FEATHERSTOM (*The Journal of Obstetrics and Gynæcology of the British Empire*, Winter Number, 1923) report the results of a series of observations they have made on the innervation of the uterus. They give details of their work which was based on clinical work and animal experiment. They summarize the present position as follows: The nervous mechanism controlling the uterus is constituted by three systems: (*a*) Local; (*b*) sympathetic; (*c*) lumbo-sacral autonomic. The "local" system is capable of producing rhythmical uterine contractions and in common with other involuntary muscle is independent of the sympathetic and autonomic systems. The sympathetic stimuli are motor to the circular muscle fibres and inhibitory to the longitudinal bundles. The lumbar cord stimuli are motor to the longitudinal fibres and inhibitory to the circular fibres. Both autonomic and sympathetic stimuli are controlled by higher centres in the medulla and possibly the cortex, but are capable of acting independently of them. Both autonomic and sympathetic reflexes are probably important factors in normal uterine contractions (compare mammary and perineal stimulation). Uterine contractions to be effective depend equally upon the integrity and correct adjustment of the balance of autonomic and sympathetic impulses. Disturbances of either, whether in the direction of augmentation or diminution, will interfere with the normal course of parturition. The authors give some practical applications of these facts. In classical Cæsarean section the advantages of lumbar narcosis, either alone or combined with a general anæsthetic, are evident. By eliminating the inhibitory stimuli from the lumbar cord and allowing the hypogastric impulses full play, the circular muscle fibres of the uterus contract firmly and in so doing reduce hæmorrhage to a negligible amount. When temporary increase of intra-uterine tension is indicated, as in *ante-partum* hæmorrhage from premature separation of a normally situated placenta, it is possible that this may be obtained without risk of inducing labour by lumbar cocainization. But in *placenta prævia* there would be greater separation and hæmrorhage would be increased. When the uterus is exhausted, spinal anæsthesia will undoubtedly diminish the tendency to *post-partum* hæmorrhage. Spinal is preferable to general anæsthesia in evacuating the uterus by the vaginal route in the earlier months of pregnancy. The authors believe that tonic uterine contraction during labour as in the "contraction ring' 'and "rigid cervix" are due to excessive sympathetic stimulation. But they hold that in normal labour spinal anæsthesia will delay the dilatation of the cervix and by diminishing the expulsive powers necessitate forceps delivery.

The Non-Protein Nitrogen and Uric Acid in Blood in Vomiting of Pregnancy.

V. J. HARDING AND KATHLEEN DREW (*The Journal of Obstetrics and Gynæcology of the British Empire*, Winter Number, 1923) have examined the non-protein nitrogen urea, uric acid and chlorides in the blood of patients vomiting during early pregnancy. Only four such observations have been recorded previously and these did not show any definite variation from the normal. They claim to show that definite disturbances exist. In a series of twenty-three patients examined they formed four groups: Mild, moderate and severe, vomiting in late pregnancy and those followed by therapeutic abortion. The treatment consisted of rest in bed with isolation and administration of glucose by the rectum or by the interstitial or intravenous route and the urging of fluids. They conclude that mild instances of vomiting of pregnancy show normal non-protein nitrogen and uric acid values in the blood. More severe forms show increased values for the blood non-protein nitrogen and uric acid. The increased values observed in the severer forms return to normal by the administration of fluids in amount sufficient to produce a diuresis. Improvement in clinical condition accompanies diuresis. When the condition was of such severity as to require therapeutic abortion extreme raised values for non-protein nitrogen and uric acid were not present. Two patients in whom prescribed treatment had failed to produce diuresis and clinical improvement did not show prompt recovery after abortion. Low urea and non-protein nitrogen ratios can be observed in all classes of cases. The chloride content of the blood in vomiting of pregnancy is normal or lowered. It usually increases on recovery.

The Influence of Pregnancy on the Wassermann Reaction and on the Clinical Manifestations of Syphilis.

FRANCIS J. BROWNE (*The Journal of Obstetrics and Gynæcology of the British Empire*, Winter Number, 1923) refers to the fact that it is not uncommon for the mothers of syphilitic infants to show no clinical signs and give no history of syphilitic infection. He undertook an investigation of one hundred syphilitic patients with a view to throwing fresh light on this matter. He concludes that in old-standing syphilis in *multiparæ* a history of infection or of primary or secondary manifestations is rarely given (2%), while in *primigravidæ* signs of syphilis are usually present at the time of examination (70%). When such evidence is not present in a *primigravida* she is usually elderly with an old-standing infection. As every *multipara* has been a *primigravida* at some period, this seems to prove that the reason why a history is not found in *multiparæ* is not that it has never been present, but that it has been forgotten or is knowingly denied. There seems to be no escape from this conclusion. A study of the Wassermann reactions in one hundred patients suffering from syphilis during pregnancy suggests that pregnancy has little or no influence in modifying the response to the Wassermann test. The Wassermann reaction on the blood from the umbilical cord is a reliable means of diagnosis of syphilis in the child. The reaction differs but little from that of the mother's blood, but on the whole tends to be slightly less positive than the latter. It is suggested that this may be due to the fact that it is purified (arterial) blood obtained from the umbilical vein.

Vulvo-Vaginitis.

IRVING F. STEIN (*Surgery, Gynecology and Obstetrics*, January, 1923) has investigated the results obtained in the causation and treatment of vulvo-vaginitis in children. He concludes that vulvo-vaginitis is an infection which is frequently gonorrhœal in origin, but may even in purulent varieties be non-specific. Filth, unquestionably plays a part in predisposing to the infection and may be the chief cause in milder forms. Diagnosis rests upon clinical evidence of the disease and smear examination made by an expert. Cultures are obtained in about 50% of gonorrhœal cases and are therefore not requisite for diagnosis. Purulent vulvo-vaginitis should be vigorously treated by an approved method such as that suggested by Gellhorn. This consists in the daily injection of an ointment of 1% silver nitrate in equal parts of vaseline and lanoline. A daily tub bath is an aid to local treatment and in mild non-gonorrhœal forms is alone required for cure. Determination of cure rests upon the clinical evidence of the infection, absence of organism on smears at intervals of one week on three occasions after suspending treatment and a period of observation equal in time to the duration of the treatment.

Operation in Cases of Collapse Following Ruptured Ectopic Gestation.

E. M. HAWKES (*Surgery, Gynecology and Obstetrics*, February, 1923) examined the clinical evidence available in one hundred and eighty-four instances of ruptured ectopic gestation to determine the value of immediate *versus* delayed operation in such cases. He concludes that deaths from hæmorrhage are not uncommon. Of eight hundred and twenty-four patients with ectopic pregnancy, one hundred and eighty-seven were prostrated from hæmorrhage. There were ten deaths from hæmorrhage in seventy-four of

these patients. The remaining one hundred and thirteen were operated on immediately. The mortality in the latter series was 8.8%, in the former 17%. These figures favour immediate operation.

NEUROLOGY.

The Pathogenesis of Cerebral Diplegia.

JAMES S. COLLIER (*Proceedings of the Royal Society of Medicine*, January, 1924) in his presidential address to the Section of Neurology opens with a few words of praise for the truthfulness of description and accuracy of deduction shown by Dr. Little, the original recorder of the disease which bears his name. He next reviews the more important contributions on causation. First, Little established the fact that diplegia is often associated with abnormalities of birth and pinned his faith to asphyxia as the cause of the lesion of the nervous system. Strictly speaking, the term "Little's disease" should be confined to diplegia which is associated with difficult birth. Actually it is used for every variety of cerebral diplegia present from the time of birth. In 1875 Erb and Charcot, ignorant of Little's work, wrongly attributed the clinical picture to a primary affection of the pyramidal tract in the spinal cord (spastic tabes in infancy). Later owing to the work of Wolters and Ross it became universally accepted that the lesion was cerebral in site—an atrophy and sclerosis of the convolutions, remarkably symmetrical, sometimes general, more often local and affecting the Rolandic region. This lesion was recognized as the end-result of some unknown previous pathological process. In 1885 Sarah McNutt, supported by William Gowers, falsely generalized meningeal hæmorrhage as the universal cause of spastic states dating from birth. Collier holds that although meningeal hæmorrhage is a common occurrence with a difficult labour, it should be deleted as a causal factor for any infantile spastic state. Encephalitis, as urged by Strümpell in 1885, is an accepted cause of infantile hemiplegia and post-natal diplegia. Fœtal encephalitis is a doubtful possibility. In 1894 Brissaud started the conception that prematurity of birth was the essential factor, inasmuch as it retarded the essential functions of evolution. To this conception there are obvious objections, the chief of which is that prematurely born children very often develope normally. Then from 1895 and onwards (Warda, Déjerine, Freud) came the conception of primary neuronic degeneration as the initial morbid process and this is the conception which Collier favours. The argument is that all diplegia dating from birth has its pathological origin *in utero* long before birth. The causative factor with the occasional exception of syphilis is entirely elusive. The cerebral paralysis resulting from difficult birth occurs when the brain is lacerated, a monoplegia or hemiplegia is the usual manifestation and the lesion is entirely different from that found in diplegia. Other ætiological factors, such as hereditary and familial influences, material ill-health during pregnancy and so on are so inconstant as to be regarded of slight importance.

Epidemic Encephalitis: Some Psychical Sequelæ.

R. D. GILLESPIE (*The Journal of Mental Science*, January, 1924) describes the psychical sequelæ in ten patients suffering from epidemic encephalitis. He says that the striking mental residuum is an emotional disorder which is most commonly an apathy. This apathy, usually pronounced, is in some instances attended by a mild degree of depression. A certain amount of euphoria may, on the other hand, be present and in one instance this amounted to definite exaltation with psycho-motor excitement. Closely connected with the emotional disorder is a volitional change; it consisted of adynamia in nine of the patients. *Flexibilitas cerea*, echolalia, mutism and rigidity may occur. Where delirium arises apart from the onset, it is probably due to a complication. There may be a slight, patchy intellectual defect. Sleep disturbance is a characteristic—hyposomnia, hypersomnia and inverted rhythm. The Parkinsonian syndrome which is such a frequent physical residuum, influences the entire prognosis unfavourably, indeed in all patients the prognosis is bad.

Brain Tumour Simulating Encephalitis.

H. L. PARKER (*Journal of Nervous and Mental Diseases*, July, 1923) records the histories of three patients observed in the Mayo Clinic in order to illustrate the difficulties in diagnosis of a tumour at the base of the brain and especially one in intricate relation with the third and fourth ventricles. Perhaps the first two conditions simulated encephalitis so closely because the two regions involved were those most commonly attacked by epidemic encephalitis. The third patient manifested a lesion sufficiently widespread and diffuse to simulate an inflammatory process, but it involved chiefly the fourth ventricle and interpeduncular fossa. General signs of tumour of the brain were sought for and not found in any of the patients. The signs present were bilateral. This is characteristic of tumours near the middle line, either in the stem, third or fourth ventricles or *corpus callosum*. Change in the cerebro-spinal fluid, elevation of temperature and leucocytosis were conspicuously absent. All the tumours were rapidly growing, soft, diffusely infiltrating and tending to induce symptoms by invasion like an inflammation, rather than by pressure. Hence the difficulty in distinguishing them from encephalitis.

Hysterical Pain.

R. G. GORDON AND H. H. CARLETON (*Brain*, July, 1923) define hysterical pain as a release phenomenon in which, owing to functional dissociation of cortical control, the more primitive sensory system convergent upon the optic thalamus is unmasked and holds sway. Its ætiology and nature have received singularly little attention either from writers on pain in general or on hysteria, yet there is no symptom so commonly complained of or so greatly considered by the patient. Hysterical headache has no pathognomonic features, but it is generally affective in character and vaguely localized, its severity depends on the attention it claims and bears no relation to the itensity of the stimulus, if indeed any stimulus can be found. In diagnosis the process of exclusion and considerations of probability must be employed. Similarly in hysterical pain in relation to scars and likewise to visceral disturbance, diagnosis can only be established by a process of exclusion. While an examination for an organic cause should be thorough it should be carried out with consonant rapidity and with the greatest care to avoid suggestion; for otherwise not only may an hysterical symptom be perpetuated, but the subjective affective element in the pain may be increased. The old search for hysterical stigmata is futile and misleading.

Hepato-Lenticular Degeneration (Wilson's Disease).

GEOFFREY HADFIELD (*Brain*, July, 1923) describes the case of a girl, aged twelve and a half years at death, who two years previously began to develope a stoop, change in facial expression, excessive salivation, speech defect and motor disability, consisting of tremor and diffuse rigidity. Attempts to use her limbs resulted in grotesque disorderly movements, in which she contracted all her muscles, but failed to accomplish the needful act. Walking was impossible. She died after a series of epileptiform convulsions. On *post mortem* examination she was found to have an intense multilobular hepatic cirrhosis. No naked eye change was seen in the lenticular nuclei, but the microscope revealed widespread degeneration of their nerve cells and neuroglial proliferation. Further, the cortex of the frontal lobes contained foci, just visible to the naked eye, in which a prolific vascular new formation, nerve fibre degeneration and a considerable degree of cell destruction were present. The author states that the case is of interest inasmuch as it fits accurately neither into the progressive lenticular degeneration group nor into that of pseudo-sclerosis, but is a transition form. In pseudo-sclerosis the muscular rigidity and destructive changes in the lenticular nuclei are minimal; in progressive lentiular degeneration, on the other hand, the rigidity is intense and the destruction of the putamina often total.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in conjunction with the MELBOURNE PÆDIATRIC SOCIETY at the Children's Hospital, Melbourne, on April 9, 1924.

The meeting took the form of a clinical meeting.

Demonstrations were conducted by the members of the honorary staff of the hospital.

Spinal Caries.

MR. W. KENT HUGHES showed a boy, aged seven and a half years, as an example of the successful treatment of tuberculous caries of the cervical spine. The measures employed comprised fixation by means of a plaster jacket, rest on a double "anterior Thomas's" splint and heliotherapy, supported by abundant fresh air and nourishing diet.

The child had first been admitted to the Children's Hospital when he was four years of age and at that time advice had been sought for him regarding stiffness and pain in the neck, then of two months' duration. A diagnosis of tuberculous caries of the third and fourth cervical vertebræ had been made by means of radiographic examination.

The boy had been fitted with a plaster jacket and discharged from hospital; he had continued to wear the jacket for nine months. At the end of this time it had been removed and the patient appeared to have improved to a very gratifying degree.

When five and a half years of age the boy had again become an in-patient in the surgical ward on account of osteo-myelitis affecting the right fibula. Radical operation, consisting of the removal of the entire shaft of the fibula had been carried out and the child had been discharged after a stay of six months in hospital.

Six months later he had returned and it had then been apparent that a retro-pharyngeal abscess had developed. Swelling had been present at the angles of the jaw on both sides of the neck and the lymphatic glands of the superior deep cervical group had been enlarged and suppurating. Free drainage had been established by incisions, but the glands had remained enlarged, firm and slightly tender. The boy had been discharged from hospital after five weeks' treatment.

Six months elapsed and it had become necessary to take the boy into hospital again as he was complaining of stiffness in the neck and a swelling had appeared on the left side. On this occasion reliance had been placed on the rest afforded by a double anterior Thomas's splint and the beneficial effects to be anticipated from heliotherapy, fresh air and nourishing food. The child's present condition was that the sinuses in the neck had ceased to discharge and he was now able to move the cervical vertebræ freely in all directions.

Cleft Palate.

DR. H. DOUGLAS STEPHENS demonstrated from eight children upon whom he had operated for the repair of cleft palate during the previous six months. He discussed the utility of the Langenbeck and Brophy operations and explained that the operation of Langenbeck was generally adopted in children between two and three years of age, while the scope of that associated with the name of Brophy, lay in the first few weeks of life and the period up to four months of age. Dr. Stephens said that he had experienced considerable difficulty in obtaining a perfect result by means of the Brophy operation and attributed this to the fact that he had hitherto seldom been able to approximate accurately the posterior portions of the hard palate at the initial operation.

Hare Lip.

Dr. Stephens also showed several infants in whom different methods of operating for single and double hare-lip were illustrated. He remarked that it was difficult to recommend any classical method; every hare-lip presented its own special features and the surgeon must be prepared to modify the operation technique to meet the conditions obtaining in the individual instance.

Paralyses.

The results of operative treatment in different types of paralyses were shown in children affected severally with cerebral birth palsy, brachial palsy and polio-myelitis.

Of two brothers one was the subject of spastic diplegia and the other of spastic paraplegia. Foerster's operation had been performed in the case of the first mentioned and the result was interesting in that although the boy had been totally unable to walk before operation with or without the aid of appliances he was able to use crutches successfully.

Dr. Stephens said that he considered the brother affected with spastic paraplegia a suitable subject for the Royle operation.

A third boy, eight years of age, had first come under observation when a week old and had then displayed a hæmatoma of the left sterno-mastoid muscle and brachial paralysis of upper arm type. Although complete recovery had taken place in all the muscles of the affected left arm the boy's parents stated that under no circumstances could he be persuaded to use the left arm when the right arm was free. Dr. Stephens had recently operated for the correction of torticollis resulting from the original injury to the sterno-mastoid muscle.

Another child was shown as illustrating an excellent result obtained by the operation of tendon transplantation in the paralysis of polio-myelitis. Before operations the patient had exhibited paralytic *talipes varus* which occasioned considerable deformity and difficulty in walking. At the time of demonstration he was able to wear boots and walked perfectly well on the flat of the foot.

Scoliosis.

Dr. Stephens discussed the treatment of scoliosis as exemplified in a girl of five years of age. He mentioned as an interesting fact that the child's mother was also a subject of scoliosis.

Polio-Myelitis.

DR. LIONEL HOOD presented two children who had recently contracted polio-myelitis. Both were boys, playmates, and were of approximately the same age, three and a half years.

The first child had become ill on March 5, 1924, and at the onset had been seized with vomiting and diarrhœa. This had continued for forty-eight hours, during which time he was stated to have been very feverish. He had seemed better on the third day, but vomiting had recurred on the day following.

On his admission to hospital on March 15, 1924, the child had exhibited great irritability and slight weakness of the muscles of the left arm. Kernig's sign had been present and an attempt at passive flexion of the neck had occasioned definite resistance. At this time the quadriceps tendon reflexes had been absent on both sides.

Cerebro-spinal fluid had been withdrawn on March 20, 1924, and sent for laboratory examination. The report received was that the fluid contained eighty-six leucocytes per cubic millimetre of which the large majority were small lymphocytes; globulin was slightly in excess, but a failure to react had attended the application of the Wassermann test.

On March 17, 1924, the weakness of the left upper limb had been noted as being more pronounced and the child's state of irritability had been unchanged. Weakness of the muscles of the right thigh had been evident on the following day and had been accompanied by great tenderness to pressure.

Since March March 24, 1924, the boy had improved, but until March 28 he had resented any attempt to flex the neck.

The second boy had become ill on March 13, 1924, six days before his admission to hospital, when he was noticed to be feverish and out of sorts. He had vomited the next day and complained of headache. Three days after the

onset of illness it had been observed that the child's power of movement of the right lower limb and in less degree the right upper limb had diminished. Control of the sphincters had also been imperfect.

On clinical examination the child had been found very irritable and to be affected with flaccid paralysis of the right lower limb. The right upper limb had been less obviously involved, but both limbs had been very tender to pressure. No tendon reflexes could be elicited. Attempts to flex the neck had occasioned muscular resistance and Kernig's sign could be detected. Since the child's admission to hospital his general condition, as also that of the paralytic limbs, had manifested great improvement.

DR. REGINALD WEBSTER gave a lantern demonstration of the micro-pathology of polio-myelitis. The slides shown reproduced micro-photographs of sections of spinal cords prepared by him during the epidemic of polio-myelitis in Victoria in 1918 and embraced both clinical and experimental polio-myelitis. The only susceptible laboratory animal was the monkey and during the epidemic referred to he had communicated the disease to monkeys.

After a brief description of experimental polio-myelitis and some remarks on the "globoid bodies," established by the Rockefeller workers as the infecting agents, Dr. Webster proceeded to show slides which demonstrated the perivascular cell "collarette," focal accumulations of round cells in both grey and white matter and ganglion cells of the anterior cornu of grey matter in successive stages of disintegration. One slide provided an example of inflammatory reaction in the posterior cornu of grey matter and another showed destructive lesions in the posterior root ganglion. It was suggested that the necrotic and reactive foci in the posterior root ganglion furnished a pathological basis for the tenderness which was frequently such a prominent feature of the disease.

Among others, a slide was exhibited to show a dense packing of the pia-arachnoid with polymorpho-nuclear and mono-nuclear leucocytes and was brought forward as accounting for those clinical types of polio-myelitis in which diagnosis from tuberculous meningitis was a matter of some difficulty.

Dr. Webster emphasized that polio-myelitis was a systemic infection which might or might not become localized in the central nervous system. It was probably not sufficiently realized that in times of epidemic the great majority of individuals who contracted the infection, did not develope paralysis. Infection was by way of the upper respiratory tract and the "carrier" played a large part in its transmission.

Tuberculous Cervical Adenitis.

DR. R. M. DOWNES, C.M.G., showed a series of eight children in whom were illustrated the results of treatment of tuberculous cervical adenitis.

In the first boy, aged thirteen years, numerous sinuses associated with caseating lymphatic glands had been present for seven years. After curettage of the sinuses the glands and some scars had been excised from the right side four weeks prior to the date of the meeting. He proposed to operate on the left side shortly and therefore had not instituted X-ray treatment.

A favourable outcome of aspiration of a softened tuberculous gland in the neck was seen in a boy of eleven years of age. The aspiration had been performed seven months previously.

A girl, aged eight years, was presented as demonstrating an apparent cure of tuberculous cervical adenitis by means of hygiene and tuberculin treatment. Indications of disease in the glands had first appeared four years previously, but the patient had been only two and a half years under treatment. Tonsillectomy had been performed.

Another girl was affected by tuberculous disease of the tonsils; her cervical glands were distinctly though not very greatly enlarged. The diagnosis of tuberculous tonsillitis was based on the extensive and classical histological lesions present in sections of the excised tonsils. The enlargement of the lymphatic glands had subsided under inunctions of 15% tuberculin.

In the fifth child shown by Dr. Downes he had performed excision of caseating glands after persevering for a long time with injections of tuberculin. Some puckering of the skin had followed the operation, but the contraction had disappeared after the adoption of massage treatment.

Excision of suppurating glands which had become adherent to the skin, had also been carried out in another patient. Puckering of the scar had again followed, but it had diminished considerably under massage.

In the case of a child in whom a discharging sinus consequent on the softening of a tuberculous gland had healed spontaneously, Dr. Downes considered that the cosmetic result would be much improved by excision of the scar.

Another child provided an example of the scar remaining after excision of a caseating tuberculous gland.

Pyloric Stenosis.

In the second instance Dr. Downes presented three infants upon whom he had performed the Rammstedt operation for the relief of congenital hypertrophic stenosis of the pylorus.

The history of the first baby was that he had vomited since birth and at the age of seven weeks had failed to gain in weight. He had been artificially fed. The vomiting had been of projectile character and associated with constipation. On physical examination peristaltic waves had been visible and a tumour in the pyloric region palpable. Some vomiting had persisted for two weeks after the operation and progress at first had been slow. It was nine months since the operation and in the interval the baby had made excellent progress.

The second baby was twelve weeks of age and operation had been undertaken three weeks prior to demonstration. He had been breast fed and had first displayed the characteristic projectile vomiting when twelve days old. In the presence of rapid loss of weight, visible peristalsis in the epigastrium and a readily palpable tumour, it had been considered advisable to perform the Rammstedt operation. The gratifying result was that the vomiting ceased and during the three weeks which had elapsed since the operation, the infant had steadily gained in weight.

A similar successful outcome of the surgical treatment of pyloric stenosis was seen in another infant, aged ten months. The operation had been performed when the baby was eight weeks old.

Perthes's Disease.

DR. WILLIAM DISMORE UPJOHN demonstrated the clinical and radiological features of Perthes's disease as exhibited by a girl of thirteen years of age. The patient, an acrobatic dancer, had suffered pain in the left hip joint for six months and had latterly been unable to walk without limping.

On the left side the greater trochanter of the femur was elevated 1.75 centimetre above that on the right side and a slight degree of flexion, accompanied by limitation of the movements of abduction and rotation was detected on the affected side. The thigh muscles on the left side did not exhibit any wasting.

By radiographic examination flattening and fragmentation of the head of the femur was disclosed and a degree of *coxa vara*. No rarefaction in the bone could be detected.

Amyotonia Congenita.

Dr. Upjohn also showed a female child as an example of *amyotomia congenita*.

Hilum Fibrosis.

DR. A. P. DERHAM supplied detailed clinical notes and radiograms of the chest of six children in order to illustrate the clinical and radiological findings in hilum fibrosis.

He said that the group had been selected almost at random from a series of some thirty patients investigated clinically and radiologically over a period of three years. In most of the children the history and symptoms had been suggestive of a chronic infection of the respiratory tract and an attempt had been made to determine the presence

or absence of pulmonary fibrosis or enlarged intra-thoracic glands before resorting to radiology. These steps had been combined in most cases with the von Pirquet test and where possible the other tests for tuberculosis.

The conclusions drawn were:

1. It is possible in a majority of cases to recognize clinically a gross amount of hilum fibrosis or enlargement of tracheo-bronchial glands, the signs being as follows: Relative dulness on percussion in the interscapular areas to a greater extent than in a normal child's chest. According to the distribution of the fibrosis this dulness extends upwards towards the apices or downwards and outwards towards the lower angles of the scapula. The dulness is accompanied over a similar area by diminished vesicular murmur with harsh breath, sounds or bronchial breathing, increased vocal resonance and sometimes by whispered pectoriloquy.

These signs have more significance when they are heard anteriorly and over areas other than the hilum and eparterial bronchus.

In most of these cases D'Espine's sign is found to the fourth or fifth dorsal vertebra or lower and sometimes there is a rosette of dilated capillaries in the interscapular area. These signs are of relative value only and may be closely simulated in very thin patients exhibiting no other signs of fibrosis.

2. X-ray screening seldom reveals the finer points of hilum fibrosis, but a satisfactory plate will show an abnormal amount of dense tissue (? fibrosis) and nodules (enlarged caseous or calcified tracheo-bronchial glands) which nearly always correspond in distribution with that suspected from clinical examinations.

3. These signs in greater or less degree are extremely common in children of all ages who have had measles, pertussis or any chronic respiratory infection, so that it is impossible to say what is normal either clinically or radiologically.

4. Some cases, which by clinical and radiological examination appeared most likely to be tuberculous proved by other other test and consideration to be probably non-tuberculous.

This last fact indicates the pulmonary fibrosis in children, even when accompanied by enlarged and apparently calcified tracheo-bronchial glands, does not necessarily indicate tuberculous infection.

Other considerations, however, indicate the probability that the condition is due to chronic infection of some sort and the indications for treatment are much the same whatever the organism at work.

5. Although chronic infection of the upper and lower respiratory tract associated with a loss of weight, chronic cough *et cetera* is common in children and is apparently a cause of pulmonary fibrosis and glandular enlargement, in only a proportion is the infection demonstrably or even probably tuberculous.

Urticaria Pigmentosa.

DR. W. W. MCLAREN AND DR. D. M. EMBELTON presented an example of *urticaria pigmentosa* as seen in a baby of eleven months of age. The child had first been brought to the children's Hospital on April 25, 1923, and had then been eleven weeks old. It had been stated by the mother that the child was born with a few spots on the hands and one large spot in the right loin. On inquiry it had been ascertained that the spots became vesicular; the vesicles which contained watery fluid did not burst, but subsided after maturation. Vesication, however, had occurred only in those present at birth and was not a feature of the lesions which appeared subsequently.

During the weeks succeeding birth crops of papules had appeared which left a mark of brown pigmentation at the points of eruption. At the age of seven month the baby had developed an eruption recognized by the mother as "hives"; at first papular, the lesions later had assumed the character of "giant urticaria" and at this time the baby had been receiving some cow's milk as supplementary to the breast milk with which he had hitherto been fed exclusively. He had been in other respect a healthy infant and had thrived very well; there had been no suspicion of syphilitic taint. He had improved to some extent since the withdrawal of the cow's milk.

Erythrœdema.

In the second instance Dr. McLaren and Dr. Embelton showed a baby, aged seven months, in whom they had made the diagnosis of erythrœdema. The child had been well until she reached the age of twelve months, but since that time she had manifested anorexia constipation, irritability and photophobia. She sweated profusely and her hands and feet were red, itchy and swollen; desquamation of the hands and feet occurred periodically. Persistent nasal catarrh was also a feature.

Various drugs, included among which were cod liver oil, extract of thyreoid gland, mercury and belladonna, had been tried by way of medication, but it could not be said that there had been any improvement in the child's condition.

On October 25, 1923, treatment by injections of stock coryza vaccine had been commenced and a steady improvement had been noted. On November 22, it had been recorded that the coryza had disappeared, the sweats had ceased and that the child was less irritable. The vaccine had been continued in gradually increasing doses until February 2, 1924, and although there had been fluctuations in the child's state of well-being, it could be said that on the whole she had made a great advance since the institution of vaccine treatment.

Diseases of the Skin.

DR. R. R. WETTENHALL showed a number of patients in whom various skin diseases were exemplified.

A child suffering from pigmented and hairy nævus had been treated with radium when eight months old. At the time of demonstration, two years later, it could be seen that there had been a complete removal of the hair and a negligible amount of pigmentation remained.

In a child suffering from angioma of the lower lip, a completely successful result of treatment by radium and X-rays was illustrated. Treatment had been carried out when the child was four months old, eighteen months prior to the date of the meeting.

A *nævus flammens* situated on the right calf had been treated by exposure to radium emanations for one hour all over the affected area. At that time the child was five months old and eighteen months had elapsed since the treatment was carried out.

The result of treatment of angioma of left ear and cheek by radium two and a half years earlier was shown. Irradiation had been maintained all over the area for two hours when the child was one month old. She was two and a half years of age.

Hæmo-lymph-angioma of right cheek in a child one year old had been treated by X-rays, filtered through aluminium. The child was two and a half years of age and the result could be said to have been completely successful.

Dr. Wettenhall presented a boy, aged six years, a native of Victoria, who exhibited skin lesions on the face, neck, forearm, hands and knees, which he considered typical of pellagra. Characteristic gastro-intestinal and nervous symptoms were also present. Dr. Wettenhall said that he intended to publish the clinical record of this boy in full at a later date.

Typical lesions of *epidermolysis bullosa* were seen in two children, one of whom had been unable to attend school for nine years.

A girl, aged eleven years, exhibited the lesions of *lichen scrofulosorum* extensively distributed on the limbs and trunk.

Tuberculous Conjunctivitis.

DR. MARK GARDNER presented a little girl, aged seventeen months, affected with tuberculous conjunctivitis. When the child was first brought to hospital the left eye had been inflamed and enlargement of the left pre-auricular gland had been noted. The glandular swelling was stated to have existed then for six weeks.

Granulations had been present on the lower palpebral conjunctiva and although the conjunctiva of the upper lid

was injected it had been free from the granular appearance exhibited by that of the lower lid.

A piece of the affected conjunctiva had been removed for pathological examination, and a report had been received from Dr. Webster that tuberculosis could be confidently diagnosed on histological grounds. From another snipping he had prepared an emulsion with which he had inoculated a rabbit and a guinea pig.

Inquiry into the family history showed that the child's grandfather had died of tuberculosis. It was stated also that the child had played for some time with a cat which had died shortly before the onset of the eye trouble.

Mongolism.

DR. J. W. GRIEVE AND DR. DOUGLAS GALBRAITH presented three children illustrating Mongolian imbecility.

The first child, aged four years, was an only child and had been born when the mother was twenty-seven years of age. The birth was said to have been difficult, but as fas as could be ascertained the baby had not needed any special measures of resuscitation after delivery.

The child's development had been very slow. She had not walked until she was three years of age and at the age of four years could say only a few words. No irregularities of dentition had been observed.

The head displayed brachycephalia and was forty-three centimetres in circumference. Features of Monogolism notable in this child included flushed cheeks, obliquely placed eyes, high arched palate and the characteristic hands in which the little fingers were contracted. A well defined systolic cardiac bruit was heard best just to the right of the sternum in the fourth intercostal space and was probably dependent on patency of the intra-ventricular septum.

Two other children were shown as presenting well defined features of Mongolism. One was the second child of two in the family, the other was an only child. The mother in each instance was forty-one years of age at the time of the birth of the child. In neither of these was there any indication of congenital heart disease, but both exhibited irregular dentition.

Splenomegaly with Jaundice.

Dr. Grieve invited discussion regarding diagnosis in the case of a girl, aged thirteen years, who eight months previously had become jaundiced. The jaundice had been of rapid development, but was not associated with much disturbance of the patient's general health. At first the stools had been clay-coloured, but they had gradually reverted to a more normal appearance. The girl was still slightly icteroid and the urine contained bile.

Physical examination revealed unequivocal enlargement of both spleen and liver.

The various measures of laboratory investigation employed included the lævulose storage test, the result of which was indicative of hepatic insufficiency. No significant changes had been observed in the routine examination of the blood and the corpuscular fragility had not been found to be raised. The Van den Bergh test applied to the blood serum had biven a bi-phasic reaction and an immediate positive result to the indirect test. The Casoni skin reaction for hydatid disease had not yielded a reaction nor had the complement fixation test with the blood serum. The blood had not reacted to the Wassermann test.

Diabetes Insipidus.

Dr. Grieve's next patient was a boy, aged eight years, who presented symptoms indicative of *diabetes insipidus*. Polydipsia and polyuria had been prominent features for the previous eighteen months. The amount of urine passed *per diem* while the child was confined to bed, had varied from two hundred and forty to four hundred and eighty cubic centimetres. Restrictions of fluid and the administration of thyreoid extract, pituitary extract and belladonna had not resulted in any improvement.

No enlargement of the *sella turcica* could be detected by X-ray examination. Estimations of the blood sugar had resulted in a normal curve. The blood urea had been determined as twelve milligrammes per hundred cubic centimetres. No reaction had been obtained when the blood serum was subjected to the Wassermann test.

Multiple Gonococcal Arthritis.

A baby of eighteen months of age had exhibited ophthalmia, the gonococcal nature of which had been demonstrated at the age of two weeks. Within a short time purulent arthritis affecting the left elbow joint, right wrist joint, both hip joints, right knee joints and tarsal joints had supervened. The joints had been treated by aspiration and gonococci had been detected in various samples of the pus withdrawn. A positive result had also been obtained when the blood serum was examined by the complement fixation test for gonococcus.

Dr. Grieve demonstrated crepitus and limitation of movement in several of the affected joints, but considered that the child had progressed very well. She was beginning to walk.

Exophthalmos.

Dr. Grieve's next patient was a girl, aged twelve years, in whom exophthalmos had been present for twelve months. The eyeballs had become rapidly more prominent during the first half of this period, but there appeared to have been no advance in the protrusion during the latter half. The right eye was more prominent than the left. Careful observation of the thyreoid gland had failed to detect enlargement at any time and no tremor had been noted. The patient's pulse rate varied between eighty and one hundred and twelve per minute, the basal metabolic rate had been determined as + 14 and the systolic and diastolic blood pressure readings were 135 and 75 millimetres of mercury respectively.

Congenital Heart Disease.

Congenital heart disease was exemplified in a boy aged four years. Cynanosis was a prominent feature and there was pronounced clubbing of the fingers and toes. The heart was enlarged and a systolic bruit was to be heard all over the præcordial area; it was of maximum intensity in the aortic region and was conducted into the neck.

Cranial Nerve Palsy.

DR. GERALD WEIGALL presented a patient who had been affected by paresis of the right facial and abducent nerves for the previous three weeks. Examination of the pupils and fundi disclosed no abnormality and there was no error of refraction.

Rheumatoid Arthritis.

Dr. Weigall's other patient exhibited swollen, tender and deformed big toes and a similar condition of the little finger of the right hand.

Radiograms, Pathological Specimens, Splints.

DR. HERBERT W. HEWLETT demonstrated from a variety of radiograms and a number of excellent colour preparations of specimens from the pathological museum of the hospital were shown by DR. B. L. STANTON.

A varied assortment of splints and orthopædic appliances was also displayed.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

GEARIN, JOHN JOSEPH, M.B., Ch.M., 1924 (Univ. Sydney), 42, Alison Road, Randwick.

Correspondence.

THE PROSTITUTION OF THE RED LAMP.

SIR: May I crave space to draw attention to the ever-increasing prostitution of the use of the red lamp. This ancient and honourable sign is being adopted, almost appropriated, by all varieties of the para- and extra-

medical professions and trades and by quacks and charlatans of every description. It is obvious that if something is not soon done, the symbol will utterly lose its significance.

It nauseates one to see it being flaunted by herbalists, "chiropracteurs," "neuropaths" and the like. One would not be much surprised if it were found in the near future adorning the beauty parlours.

Would it not be desirable to attempt to have its use restricted by law to legally qualified medical practitioners and qualified pharmacists?—not less in the interests of the public than those of the physician and pharmacist. I think also that our colleagues of the dental profession (certainly the upper strata of them) will admit that if they need a distinguishing lamp it might very well be of some other colour, for example, orange.

The red lamp ought to indicate nothing more and nothing less than that relief from physical ills and particularly physical emergencies may be obtained where it is exhibited.

Yours, etc.,

GILBERT G. BRADLEY.

Northbridge,
April 22, 1924.

Proceedings of the Australian Medical Boards.

VICTORIA.

THE following alterations have been registered, under the provisions of the *Medical Act, 1915*:

Name of Practitioner Changed in the Register.

HENDERSON, MARY ANKETELL to BELL, MARY ANKETELL.

Additional Diplomas Registered.

JACOBS, HUBERT SYDNEY, Dip. G.O., 1922 (Dublin); F.R.C.S., 1923 (Edin.).

Names of Deceased Practitioners Removed from the Register.

ESLER, ALFRED WILLIAM.

McDOUGALL, RICHARD.

O'BRIEN, JOHN ALOYSIUS.

RINDER, ALFRED WILLIAM.

QUEENSLAND.

THE undermentioned have been registered, under the provisions of the *Medical Act of 1867*, as duly qualified medical practitioners:

BLAIR, JOHN MURRAY, M.B., B.S., 1923 (Univ. Melbourne), Mitchell.

FLYNN, THOMAS JOSEPH, M.B., Ch.M., 1922 (Univ. Sydney), Ipswich.

HUXTABLE, CHARLES REGINALD RALSTON, M.B., 1915 (Univ. Sydney); F.R.C.S., 1922 (Edin.).

SHANASY, FRANCES LILLIAN, M.B., B.S., 1921 (Univ. Melbourne), Mitchell.

SHIEL, DOMINIC VICTOR, M.B., B.S., 1923 (Univ. Melbourne), Children's Hospital, Brisbane.

Medical Appointments Vacant, etc..

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii..

PUBLIC SERVICE BOARD, N.S.W.: Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Society's Dispensary Friendly Society Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 12, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

MAY 7.—Victorian Branch, B.M.A.: Branch.
MAY 8.—Brisbane Hospital for Sick Children: Clinical Meeting.
MAY 9.—Queensland Branch, B.M.A.: Council.
MAY 9.—South Australian Branch, B.M.A.: Council.
MAY 14.—Tasmanian Branch, B.M.A.: Branch.
MAY 14.—Melbourne Pædiatric Society.
MAY 21.—Victorian Branch, B.M.A.: Council; Election of Representative on Representative Body.
MAY 21.—Western Australian Branch, B.M.A.: Branch.
MAY 23.—Queensland Branch, B.M.A.: Council.
MAY 29.—South Australian Branch, B.M.A.: Listerian Oration.
JUNE 4.—Victorian Branch, B.M.A.: Branch.
JUNE 6.—Queensland Branch, B.M.A.: Branch.
JUNE 11.—Melbourne Pædiatric Society.
JUNE 11.—Tasmanian Branch, B.M.A.: Branch.
JUNE 12.—Brisbane Hospital for Sick Children: Clinical Meeting.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, B.M.A. Building, 30-34, Elizabeth Street, Sydney. (Telephone: B. 4635.)

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